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A HANDBOOK
OF
PATHOLOGICAL ANATOMY
AND
HISTOLOGY

WITH AN INTRODUCTORY SECTION ON
POST-MORTEM EXAMINATIONS
AND
THE METHODS OF PRESERVING AND EXAMINING DISEASED TISSUES

BY
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PREFACE

TO THE THIRD EDITION.

In the third edition of this work, the same objects have been kept in view as in the second edition.

It is intended that the student and the practitioner shall find in it the information which they need to enable them to perform autopsies, to preserve the tissues, to prepare them properly, and to examine them with the microscope.

The work, therefore, comprises instruction in the methods of making post-mortem examinations, of preserving diseased tissues, of preparing them for microscopical examination, and of cultivating and examining bacteria. It also gives an account of the lesions of the different parts of the body, of the general diseases, of violent deaths, and of poisoning; of the changes produced by inflammation and degeneration; and of the structure of tumors.

All of the drawings have been made by the authors.

FRANCIS DELAFIELD,
T. MITCHELL PRUDDEN.

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PART I.

THE METHOD OF MAKING
POST-MORTEM EXAMINATIONS
AND OF
PRESERVING DISEASED TISSUES.

THE METHOD

OF MAKING

POST-MORTEM EXAMINATIONS.

THE particular object of making a post-mortem examination varies in different cases. It may be to determine whether a person has died from violence or poisoning; to account for a sudden death; or to study the lesions of disease. In any case the examination should embrace all the important parts of the body, not merely a suspected organ, and it should be recorded at the time it is made.

In endeavoring to ascertain the cause of death, when the clinical history is imperfect or unknown, great care is necessary. Mechanical causes of death, which destroy life by abolishing the function of one of the important viscera, only occur in a moderate number of cases. Most of the lesions which we find after death rather indicate the disease than the cause of death. We do not know how great a degree of meningitis, or of pneumonia, or of endocarditis, or of cirrhosis, or of nephritis, necessarily causes death. On the contrary, we find that one patient recovers with an extent of lesion that is sufficient to destroy the life of another. So with accidents; there is often no evident reason why a fracture of the skull or of the pelvis should destroy life, but yet they usually do. In some of the general diseases, such as typhoid fever, the lesions cannot always be called the cause of death; in others, such as typhus fever, there may be no evident lesions at all. Sudden deaths of persons who have apparently been in good health up to the time of death are often particularly obscure. In many of them, we have to acknowledge that we can find no sufficient cause for the death. This is of course due to our imperfect knowledge. But it is much better in such cases to avow our ignorance than to attribute the death to some trifling lesion. The brain and the heart are the organs which are especially capable of giving symptoms during life without corresponding lesions after death. Very well-marked cardiac or cerebral symptoms may continue for days months, and apparently destroy life, and yet after death we find no corresponding anatomical changes. It is the novice in post-mortem examinations who is particularly apt to mistake ordinary post-mortem appearances for lesions.

EXTERNAL INSPECTION.

Before commencing the examination of the internal viscera, it is always necessary to make some inspection of the external surface of the body. The minuteness of this inspection will depend upon the character of the case: in the case of an unknown person, or of one suspected to have died from unnatural causes, it is necessary to search for and record, not only all contusions, wounds, etc., their size, situation, and condition, but also deformities from disease and any physical peculiarities of hair, eyes, teeth, moles, etc., by which the person may be identified. In such cases it is well, if possible, to have a photograph taken of the entire body. In cases of doubtful identity, it is sometimes wise to make a wax or plaster cast of the outside of the teeth and jaws. In ordinary examinations, we note the general nutritive condition of the body, and look for evidences of external injury, for skin diseases, ulcers, œdema, gouty deposits, abscesses, enlarged lymphatic glands, etc. The glans penis and prepuce are to be carefully searched for syphilitic cicatrices.

It is usual to find certain changes in the external appearances of the body which are due to the cessation of life and the commencement of decomposition. We speak now of bodies which have not been buried, but which have been kept in the ordinary way, lying on the back and loosely covered with a shroud, or dressed with the ordinary clothes.

Cadaveric Lividity.—After life becomes extinct and before the blood coagulates it settles in the veins of the more dependent parts of the body, producing, usually within a few hours after death, a mottling of the surface with irregular livid patches. These patches may coalesce, forming a uniform dusky red color over the back of the trunk, head, and extremities, and sometimes over the ears, face, and neck. The same effect is observed on the anterior aspect of the body if it has lain on the face. At points of pressure, from folds in the clothes or from the weight of the body on the table, the red color is absent or less marked. These changes occur before putrefaction sets in. This cadaveric lividity or hypostasis should not be mistaken for ante-mortem ecchymosis, from which it may usually be readily distinguished by its position and extent, by the fact that the surface of the skin is not elevated, and by the fact that on incision no blood is found free in the interstices of the tissues. Not infrequently the subcutaneous tissue in the vicinity of these post-mortem hypostases becomes infiltrated with reddish serum. Very soon after death, particularly in warm weather, the tissues immediately around the subcutaneous veins of the neck and thorax and in other situations may become stained of a bluish-red color from the decomposition and escape from the vessels of the coloring matter of the blood. If the epidermis has been detached at any point, the skin beneath soon becomes dry and brown.

Putrefactive Changes.—Usually in from one to three days, depending upon circumstances, a greenish discoloration of the skin appears, at first upon the middle of the abdomen, over which it gradually spreads, assuming a deeper hue and often changing to a greenish purple or brown. Greenish patches may now appear on different parts of the body, earliest upon those overlying the internal cavities; this discoloration is probably produced by the action on the hæmoglobin of gases developed by decomposition. The eyeballs now become flaccid, and if the eyelids are not closed the conjunctiva and cornea become brown and dry. The pressure of gases developed by decomposition in the internal cavities not infrequently forces a greater or less quantity of frothy, reddish fluid or mucus from the mouth and nostrils, producing distention of the abdomen, and, if excessive, may produce changes of position of the blood in the vessels, and even a moderate amount of displacement of the internal organs.

After five or six days, under ordinary circumstances, the entire surface is discolored green or brown. After this the epidermis becomes loosened from the formation of gases and separation of fluids beneath, and the tissues become flaccid. The abdomen and thorax may be greatly distended, the features distorted and scarcely recognizable from swelling, and the hair and nails loosened. Beyond this stage of putrefaction, the consecutive changes, leading to more or less disintegration of the soft tissues, can scarcely be followed with certainty. The rapidity with which these changes follow one another depends upon a variety of conditions, such as temperature, moisture, access of air, and the diseases which have preceded or caused death. Thus an elevated temperature, and the presence of air and moisture, hasten the advent and progress of putrefactive changes. The bodies of infants usually decompose more rapidly than those of adults, fat bodies more quickly than lean ones. The infectious diseases, intemperance, and the puerperal condition promote rapid decomposition; as does also death from suffocating gases. Poisoning by arsenic, alcohol, antimony, sulphuric acid, strychnine, and chloroform may retard the progress of decomposition. Burial in dry soil and submersion in water also retard the progress of decay.

Cooling of the Body.—The internal temperature of the healthy living body is about 37.5° C. (99.5° Fahr.). But it may be increased several degrees in consequence of disease. After death, the chemical changes upon which the maintenance of this temperature depends, rapidly diminish, and the body gradually cools to the temperature of the surrounding medium. This usually occurs in from about fifteen to twenty hours, but the time required depends upon a variety of conditions. Immediately after death, there is, in nearly all cases, a slight elevation of internal temperature, owing to the fact that the metabolic changes in the tissues still continue for a time, and the blood ceases to be cooled by passing through the lungs and peripheral capillaries. After death from certain diseases, yellow fever, cholera, rheumatic fever, and tetanus, a considerable elevation of internal temperature has been repeatedly observed. The time occupied by the cooling of the body may be prolonged after sudden death from accidents, acute diseases, apoplexy, and asphyxia. A number of cases are recorded in which the body retained its heat for several days without known cause.

After death from wasting chronic disease, and in some cases after severe hæmorrhages, the cooling of the body is very rapid, the external temperature being reduced to that of the surrounding air within four or five hours. Fat bodies cool less quickly than lean ones, the bodies of well-nourished adults less quickly than those of children or old persons. The temperature of the surrounding medium, the degree of protection of the body from currents of air, will, of course, modify the progress of cooling; and the internal organs retain their heat naturally longer than the surface of the body. The rate at which cooling occurs is most rapid, as a rule, during the hours immediately following death, notwithstanding the post-mortem rise which may ensue.

It will thus be seen that if required to pronounce upon the time which has elapsed since death in a given case, we can do so only approximately. It is necessary to take into account all of the above-mentioned conditions which modify the rate of cooling of the body, and then we may be able to state only the probabilities of the case. It is furthermore unsafe in any case to infer the cause of death from the rate of cooling of the body.

Rigor Mortis.—Death is usually succeeded immediately by a period of complete muscular relaxation; the jaw drops and the limbs become flaccid. The muscles may retain for two or three hours, however, the capacity of contracting on the application of appropriate stimuli. On the average within six hours the muscles become firm and rigid. This post-mortem rigidity is called *rigor mortis.* On the occurrence of the rigor mortis, the muscles become fixed in whatever position they may have had at the time of its occurrence. It usually begins in the muscles of the eyelids, extends to those of the back of the neck and lower jaw, then to the face and neck, and thence passing downward affects the muscles of the thorax and lower extremities. It usually disappears in the same order. Although commencing on the average six hours after death, it may set in almost instantly, or it may be delayed for twenty-four hours or more. It may pass off very rapidly, in rare cases in from one to three hours; or it may persist for two or three weeks or longer. It may be said in general that the average time of its disappearance is within twenty-four or forty-eight hours after its occurrence, depending on temperature, its intensity, the mode of death, the period of its advent, etc. Caspar states that in fœtuses before term he has never observed rigidity, and that in young children it is feeble and of short duration. Its occurrence and phenomena may be in some cases of the highest medico-legal importance; but its careful observation does not, with our present knowledge of its significance, appear to essentially further the aims of the practical pathologist.[1]

Contusions.—It is often important to determine whether violence has been inflicted upon a body before death. In regard to this point, we must remember, first, that blows and falls of sufficient violence to fracture bones and rupture the viscera may leave no marks on the skin, even though the person has survived for several days; and, second, that there are post-mortem appearances which simulate ante-mortem bruises. A severe contusion during life may present, at first, no mark or only a general redness. After a short time, the injured part becomes swollen and of a red color; this color may be succeeded by a dark blue, and this in turn fade into a greenish yellow or yellow; these later appearances are due to an escape of blood from the vessels and to a subsequent decomposition of hæmoglobin. If therefore we cut into such an ecchymosis after death, we find extravasated blood or the coloring matter of the blood, in the form of pigment granules, free in the tissues. Post-mortem discolorations, on the other hand, although their external appearance may resemble that of ante-mortem ecchymosis, are not formed by an extravasation of blood, but by a circumscribed congestion of the vessels, or by an escape of blood-stained serum. If we cut into such discolorations, therefore, we find no blood outside the vessels.

Blows on the skin of a body which has not been dead for more than about two hours may produce true ecchymoses with extravasation of blood, such as can be distinguished with great difficulty or not at all from those formed during life. If putrefactive changes be present, the difficulty of distinguishing between ante-mortem and post-mortem bruises is greatly enhanced.

Hanging and strangulation are attended with the formation of marks on the neck which are fully described in works on forensic medicine. These marks must not be confounded with the natural creases of the skin of the neck. Many adults during life have creases of the skin of the neck, one or more in number, running downward from the ear under the chin or encircling the neck. After death, these creases may be much more evident than during life, and may be rendered more decided by the position of the head and the freezing of the body. They regularly persist until the skin putrefies.

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[1] For further details concerning rigor mortis, putrefactive changes, particularly the later stages, and the phenomena of cooling of the body, see *Tidy*, "Legal Medicine," vol. i., pp. 52–120, or other works on medical jurisprudence.

Wounds.—We should notice the situation, extent, and direction of a wound; the condition of its edges and the surrounding tissues. If it be a deep, penetrating wound, its course and extent should be ascertained by careful dissection rather than by use of a probe.

If the edges of a wound be inflamed and suppurating, or commencing to cicatrize, it must have been inflicted some time before death. In a wound inflicted a short time before death, the edges are usually everted; there may be more or less extravasation of blood into the surrounding tissues, and the vessels contain coagula; but sometimes none of these changes are observed. The chief characteristics of a wound inflicted after death are, absence of a considerable amount of bleeding, non-retraction of the edges, and the absence of extravasation of blood into the tissues. But a wound inflicted within two hours after death may resemble very closely one inflicted during life. In general, unless a wound is old enough for its edges to present inflammatory changes, we must be very careful in asserting its ante-mortem or post-mortem character.

Fractures.—It may be important to determine whether a bone was fractured before or after death. This point cannot always be decided. Fractures inflicted during life are, as a rule, attended with more extravasation of blood and evidences of reaction in the surrounding tissues; but fractures produced within a few hours after death may resemble these very closely. Usually a greater degree of force is necessary to fracture bones in the dead than in the living body.

Scars and Tattoo Marks.—The presence and character of cicatrices should be noticed. Scars produced by any considerable loss of substance may become very much smaller and less conspicuous, but never entirely disappear. Slight and superficial wounds, however, produce marks which may not be permanent. The discoloration produced by tattooing may, although rarely, disappear during life.

INTERNAL EXAMINATION.

After completing the external inspection of the body, we commence the internal examination. In order that this examination may be made both thoroughly and rapidly, we should follow a regular method. The method should be such as will enable us to examine the relations of parts to one another, without seriously disturbing them, and to remove and inspect the organs in such an order and manner as will not interfere with the examination of parts which are to follow. In certain cases it may be necessary to depart from the regular method; but, as a rule, the following plan will be found most advantageous.

It is important to remember the difference between the distribution of the blood in the body during life and after death. During life, the blood is in constant motion and is distributed in a regular way in the heart, capillaries, arteries, and veins. Inflammations and obstructions to the circulation may disturb this natural distribution and produce congestion of particular parts of the body. After death, the blood ceases to circulate; it leaves the left cavities of the heart, the arteries and capillaries, and collects in the veins and the right cavities of the heart. According to the character of the disease which causes death, coagulation of the blood takes place more or less extensively and at an earlier or later period. The local congestions which existed during life often disappear after death. On the other hand, local congestions are found after death which did not exist during life. Thus, after death, the scalp often contains a large amount of venous blood. The veins of the pia mater and the sinuses of the dura mater may be filled with blood. The mucous membrane of the larynx and trachea may appear to be deeply congested. The lungs are congested if the patient has been comatose for some hours before death. All the tissues of the back and the membranes of the spinal cord are often gorged with venous blood. The right auricle and ventricle of the heart may contain fluid or clotted blood in considerable quantity.

THE HEAD.

The scalp is divided by an incision across the vertex from ear to ear. The flaps are dissected forward and backward, taking up the temporal muscles with the skin, and leaving the pericranium attached to the bone. The internal surface of the scalp and the pericranium are to be searched for ecchymoses and inflammatory lesions. A circular incision is then made with a saw, and the roof of the cranium removed. The incision in front should pass through a point about three and one-half inches above the bridge of the nose, behind through the occipital protuberance. When the roof of the cranium is thus entirely loosened, a stout hook is introduced under the upper edge of the calvarium, and this is wrenched off with a jerk.

Sometimes the dura mater is so adherent to the calvarium that the latter cannot be torn from it without injury to the brain. In this case, the dura mater may be cut through at the level of the cranial incision, and the brain removed with the calvarium and separated afterwards. Or, which is better, after the circular incision has been made through the bone, a longitudinal incision is made, from front to back, about three-quarters of an inch to one side of the median line of the skull, and a segment of bone removed. The knife blade may now be inserted from the open side, and the dura cut away from the skull cap along the line of the longitudinal sinus, where the adhesions are apt to be most firm.

We should notice whether or not the calvarium is symmetrical. The cranial bones increase in size by a growth of bone at the edges of the sutures. If any suture become completely ossified and closed prematurely, the bones will be unequally developed. The thickness and density of the cranial bones vary considerably within the limits of health. There are often deep depressions on the inner surface of the skull along the sagittal suture, caused by the pressure of the Pacchionian bodies, and of no pathological significance. We should observe the blood content of the bone, determine the existence or absence of fractures, inflammatory lesions, exostoses, etc.

The Dura Mater is now exposed to view. It will be found more or less adherent to the calvarium; a moderate amount of adherence, especially in old persons, does not denote disease. Very extensive and firm adhesions are usually produced by inflammation. Near the median line, the Pacchionian bodies often project through the dura mater and may produce indentations in the internal surface of the calvarium. We must look for clots and for tumors and for inflammatory lesions on the external surface of the dura mater. The longitudinal sinus should be laid open and its contents examined. A circular incision is then made through the dura mater in a line corresponding to the cranial incision; the falx is divided between the anterior lobes of the brain, and the entire membrane drawn back. We should observe the existence of abnormal adhesions of the dura mater to the pia mater, bearing in mind that a moderate amount of adhesion along the longitudinal fissure is normal. The internal surface of the dura mater is to be examined for the products of inflammation and for tumors.

The Pia Mater covering the convex surface of the brain is now exposed. The degree of congestion, and the existence of serum, pus, or blood, beneath, within, or upon it, are now to be ascertained before the brain is removed. The pia mater in old persons frequently loses its transparency and becomes thick and white; this change is most marked along the longitudinal fissure and large vessels. Marked and general thickening of the pia mater is produced by chronic inflammation. Along the longitudinal fissure, and sometimes at a considerable distance from it, we usually find small elevated whitish nodules, which are the *Pacchionian bodies* and are normal in the adult.

The amount of serum beneath the pia mater varies. A considerable amount, especially in cachectic persons, may exist without brain disease. Clear serum raising the pia mater and separating the convolutions of the brain may be simply dropsical or due to chronic meningitis. Turbid and purulent serum, beneath and in the pia mater, is produced by acute or chronic meningitis. The degree of flatness of the surface of the convolutions should be observed before removing the brain; for, when marked, it affords an important indication of pressure, from hæmorrhage, inflammatory products, internal fluid effusions, and tumors. The pia mater should be carefully examined for miliary tubercles.

The Brain.—After examining the convex surface of the brain, the anterior lobes of the cerebrum are to be pulled gently backward, the nerves, vessels, and tentorium severed, and the medulla cut across as low down as possible. The brain is now removed from the cranium by passing the fingers of one hand down, beneath and behind the lobes of the cerebellum, and drawing the brain out, supporting the convexity with the other hand.

The adult brain in the male weighs on the average about 1,400 gm. (49½ oz.); that of the female, about 155 gm. (5 oz.) less. The average proportional weight of the brain to that of the body is about one-forty-fifth, although in this, as in the absolute weight, there is considerable variation.

The exact situation of any lesion which is apparent externally, should be described by its relation to the lobes, fissures, convolutions, and sulci.

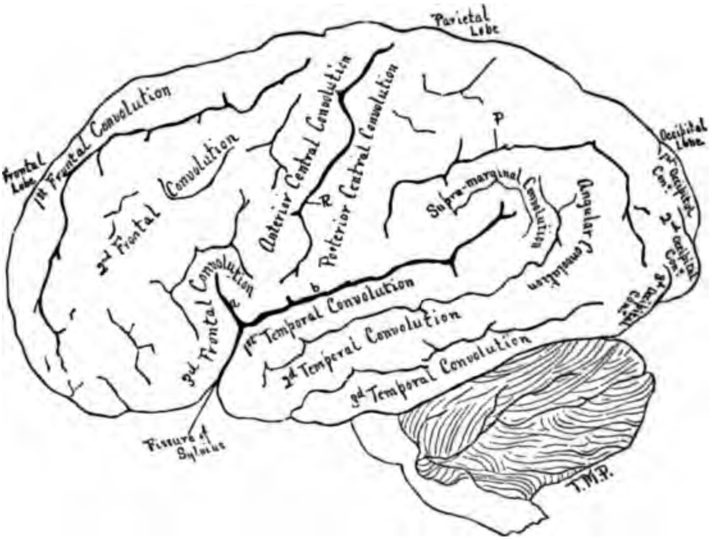

FIG. 1.—SIDE VIEW OF THE HUMAN BRAIN, SHOWING ITS FISSURES AND CONVOLUTIONS.

The brain is first laid upon its convex surface, and the anterior, middle, and posterior cerebral arteries, as well as the basilar and the carotids, are to be examined for emboli, thrombi, atheroma, and aneurisms. Evidence of extravasations of blood, tumors, and inflammatory lesions are now to be looked for. The brain is next turned over on to its base. An incision is made through the pia mater over the convex surface of the cerebrum. The membrane is stripped up, and its adherence to the brain and its thickness noted.

The more common method of opening the brain is as follows: the halves of the cerebrum are to be separated until the superior surface of the corpus callosum is exposed. A longitudinal incision is made through the junction of the corpus callosum and the cerebrum, and downward into the ventricle. The incision should be made carefully, so as not to cut through the ventricle into the ganglia below. The incision thus made through the roof of the ventricle is prolonged backward and forward in the direction of the cornua, so as to expose the entire ventricle. A longitudinal incision is then made outward and backward into the hemisphere, from the outer edge of the lateral ventricle, nearly to the pia mater. A second incision is then made through this cut surface outward, and this is repeated until the hemisphere is divided into a number of long, prism-shaped pieces, held together by the pia mater and a small portion of the cortex. The brain is now turned around so as to bring the other hemisphere under the hand, and the operation is repeated on the other side.

The size, shape, and contents of the ventricles should be noticed, and the thickness and appearance of the ependyma.

The fornix and the central portion of the corpus callosum are cut across by passing the point of the knife through the foramen of Munro and cutting upward. They are then drawn backward, one of the posterior cornua of the fornix being severed and laid to one side. The velum interpositum and the choroid plexus are now dissected up, the blood contents and the general appearance noted, and the third ventricle examined. Not infrequently small cysts of the choroid are found, which seem to have little or no pathological significance.

The fourth ventricle is now opened by a longitudinal incision through the vermiform process. Each hemisphere of the cerebellum is divided first into two parts by an incision through the upper and inner convex border, and then each segment is further divided by incisions in the same direction.

Thin transverse sections are now made through the cerebral ganglia, commencing in front. The ganglia are supported, and the sections caused to fall apart as they are cut, by carrying the fingers of one hand under the brain, and gently lifting the ganglia at points just beneath where the sections are made. It is important to observe the exact position of any lesion which may be discovered in the cerebral ganglia, their relations to the external and internal capsule, and to the caudate and lenticular nucleus.

Finally the segments of the cerebrum and cerebellum are folded up together into their original positions, the whole is turned over on to the vertex, and thin sections are made through the medulla. Small clots in the medulla should not be overlooked.

In case of the discovery of apoplectic clots, areas of softening, etc., either in the hemispheres or in the basal ganglia, after their location and extent is determined, they should be carefully searched for lesions of the blood-vessels, minute aneurisms, areas of degeneration, and ruptures. For this purpose it may be necessary to allow a stream of water to run over the affected portion, so as to wash out the brain substance and expose the vessels. In some cases the blood-vessels are best exposed by macerating the brain tissue at the seat of the lesion for some hours in water, and then washing out the brain substance under the faucet.

While the above mode of dissecting the brain gives a very complete view of the seat and extent of lesions in general, where a more exact localization of lesions with a microscopical examination is to be made the following is a better method of opening the brain:

After completing the external examination, as detailed above, the brain is laid on its vertex, the cerebellar end towards the operator. The cerebellum is raised by the fingers of the left hand, and the pia cut through along the sides of the corpora quadrigemina and along the inner margin of the temporal lobes to the middle cerebral artery on both sides. Then, raising the temporal lobes, in turn, by their apices, the pia is cut through along the course of the middle cerebral artery into the Sylvian fissure, and along the course of its posterior branch to its end. Now drawing the temporal lobes one after the other upward and outward, their junction with the base is cut, the knife being held horizontally so as not to injure the basal ganglia, until the descending horn is opened. The point of the knife being in the descending cornua, the incision through the brain substance then passes outward and backward well into the posterior cornua, thus partially severing, at the lateral surface of the brain, the junction of the occipital and temporal lobes. The temporal lobes are then turned outward and backward (Fig. 2).

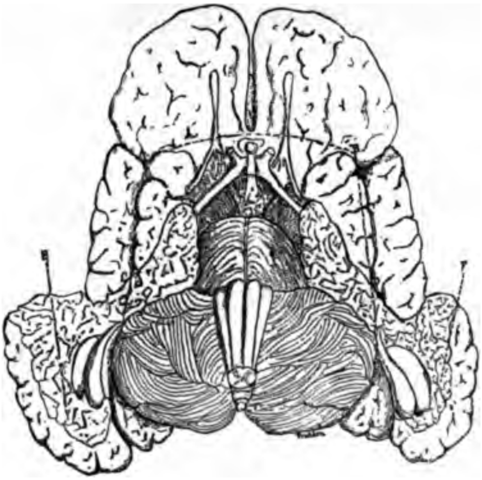

FIG. 2.—VIEW OF THE BASE OF THE BRAIN, WITH THE TEMPORAL LOBES TURNED BACKWARD AND OUTWARD.
A, B. Line of transverse incision.

The operculum is now pulled well outward, completely exposing the island of Reil, and a slightly curved transverse incision is made, deep enough to pass into the anterior horns of the ventricles, connecting the anterior sulci of the island of Reil (Fig. 2, A, B).

Now raising the cerebellum and inserting the point of the knife into the ventricle, with short incisions from within outward cut through the internal capsule on either side from back to front, care being taken not to injure the basal ganglia. Then cut across the crura of the fornix and the septum lucidum, leaving the fornix lying on the corpus callosum.

The square basal piece thus freed—*the brain axis*—includes the island of Reil, the basal ganglia, the crura, pons, medulla, and cerebellum (Fig. 3).

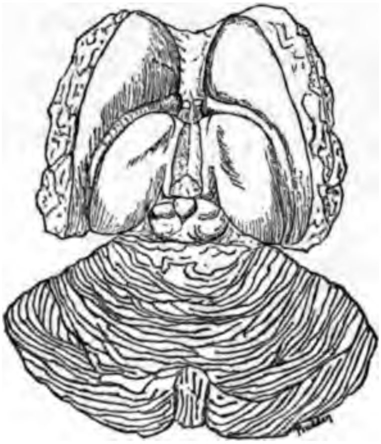

FIG. 3.—DRAWING OF THE BRAIN AXIS, SEPARATED FROM THE BRAIN MANTLE, AS SEEN FROM ABOVE.

The remaining portion—*the brain mantle*—includes the convolutions, corpus callosum, and fornix.

The basal piece may be further examined by a series of transverse incisions from one-half to three-quarters of an inch apart, and it may then be hardened either with or without the cerebellum. The convolutions may be cut into small pieces by longitudinal and transverse incisions, made from within and not reaching quite to the pia mater, which will then serve to hold the pieces together in their proper relations to one another.[1]

For the methods of hardening and preserving the brain tissues, see p. 15.

The Base of the Cranium.—We now return to the skull. The remaining sinuses of the dura mater should be opened, and this membrane then entirely stripped from the bone. The bones at the base of the skull are to be examined for fractures, inflammatory lesions, and tumors. In cases of acute purulent meningitis, the temporal and frontal bones should be carefully examined, as the inflammatory process is sometimes transmitted from the internal ear, or mastoid cells, or frontal sinuses.

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[1] For further details of this method of opening the brain and a consideration of its advantages, see *Van Gieson*, New York Medical Journal, July 20th, 1889.

The eyes may be removed by breaking the roof of the orbit with a hammer, removing the fragments of bone, and dissecting away bone and muscles, so as to expose the optic nerve and posterior segment of the eye. That portion of the globe which is not covered by conjunctiva can now be cut away with scissors, and removed with the optic nerve, or, when permissible, the whole eye may be cut out.

Hardening and Preservation of the Tissues for Microscopical Examination.—For the study of tumors and inflammatory lesions of the bones of the skull and ossifications of the dura mater and pia mater, the affected portions should be cut into small pieces, decalcified with picric acid, and subsequently hardened in strong alcohol. In the ordinary lesions of the dura mater, the tissues are best hardened and preserved by stretching the diseased portions on a flat piece of wood or cork with pins, and placing them in Müller's fluid.[1]

The pia mater is so delicate that if it be torn from the brain when quite fresh, its tissues are apt to be injured. The portions of the pia mater which are to be preserved should therefore be removed by cutting off slices of the brain substance about one-half an inch thick, with the membrane still attached, and placing the whole in Müller's fluid. After twenty-four hours the pia mater will have become sufficiently hard to permit of its being stripped off without injury, and it is then spread loosely on a flat cork with pins, the free surface outward, and the cork floated, specimen side down, in a dish of alcohol (80 per cent). The next day strong alcohol may be used, and the hardening is complete in three or four days. The pia mater should not remain longer than twenty-four hours in Müller's fluid before being stripped off; for after this time it usually becomes so firmly attached to the brain substance as to render its removal very difficult.

The ependyma, when this alone is to be studied, may be sliced off with a sufficient quantity of underlying brain substance to prevent its folding, and hardened in Müller's fluid. Special care should be exercised not to touch the surface of the ependyma, since the epithelium is easily rubbed off.

The brain substance, after having been cut into sufficiently small pieces for the general examination, should be hardened in Müller's fluid. Large quantities of the fluid should be used, four or five times the bulk of the tissue, and the pieces of tissue should either be suspended in gauze or kept apart by a little absorbent cotton. The fluid should be kept cool, and changed on the second, fifth, and eighth days, and again in the third week.

In general, two or three months are required to secure a good hardening with Müller's fluid. When the hardening is complete, the brain tissue is rinsed off with water and put in eighty-per-cent alcohol, in which it may be kept, preferably in the dark.

Great care is required in hardening and preserving nerve tissues, and most of the ordinarily practised methods give only caricatures and gross distortions of the brain structure.

Too long a soaking in Müller's fluid renders brain tissue very brittle. To get the best hardening, the single pieces ought not to be larger than 1 c.c.

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[1] For the details of the methods of hardening, decalcifying, staining, etc., see the end of Part I.

Certain lesions, particularly the softenings of the brain, are best studied by teasing, when fresh, in one-half-per-cent solution of sodium chloride, or in frozen sections of the fresh tissue. (See chapter on the Nervous System.) The blood-vessels should be stretched on cork with pins, and hardened with Müller's fluid and alcohol. The eye and portion of the optic nerve, if removed, should be hardened with Müller's fluid.[1]

THE SPINAL CORD.

The examination of the spinal cord is usually most conveniently made after the removal of the brain.

The body should be placed face downwards, with a block under the thorax and the head hanging over the edge of the table. An incision is made through the skin and muscles along the entire length of the spine, and the soft parts dissected away on each side so as to expose the laminæ of the vertebral column. The laminæ are then divided close within the articular processes, with the rachitome or with the saw and chisel. The spinous processes and laminæ are then torn away together with a stout hook, and the cord is thus exposed.

The saw should be so directed in severing the laminæ that the incision shall touch the outer border of the spinal canal, as otherwise the laminæ and spinous processes are not easily removed.

By means of a long, curved chisel, made for this purpose, the bodies of the vertebræ may be removed from the front after the thoracic and abdominal viscera are taken out, and the cord thus exposed and removed.

When the body has lain on the back, the membranes of the cord may be found considerably congested without indicating the pre-existence of any disease. If the body has lain for some time, especially in warm weather, serous fluid may have accumulated within the membranes as a result of post-mortem change.

The roots of the nerves are now to be cut across, as far away as possible from the cord, and the cord removed in its membranes, care being taken not to press it in any way. It is the safest plan not to grasp the cord itself, but with a forceps to seize the dura mater and thus lift it up at once as it is freed from its attachments. It is now laid on the table, and the dura mater laid open with scissors on the anterior and posterior surfaces over its entire length, and searched for tumors, inflammatory lesions, etc. The finger should be passed gently along the cord as it lies on the table, so as to detect any marked softening or sclerosis. It should now be held lightly over the fingers, and smooth transverse incisions made, with a very sharp knife, about one-half an inch apart through its entire substance, leaving the segments attached to the pia mater. The cut surfaces should be carefully examined for abnormal blood contents, hæmorrhages, inflammatory lesions, softening, scleroses, and pigmentations. Important lesion of the cord may be invisible to the naked eye, and hence, if disease be suspected, the organ should be preserved for microscopical examination. After removal of the cord, fractures and displacements of the vertebræ are easily recognized.

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[1] For further details of the methods of dissecting, hardening, and preserving the brain and spinal cord, consult *Van Gieson, loc. cit.*

Preservation of the Spinal Cord and its Membranes, and of Peripheral Nerves.—The entire cord, with its nerve roots and dura—the segments into which it has been cut for gross examination being left in place—should be laid or suspended in a large jar of Müller's fluid, the segments slightly separated from each other by a little absorbent cotton.

The hardening and preservation of the cord should be done by the same method as suggested above for the brain. The same care should be exercised as in the brain not to permit the cord to become brittle by remaining too long in the Müller's fluid. If the dura mater of the cord alone is to be preserved, it should be treated in the manner suggested for the dura mater cerebralis. The pia mater spinalis is best studied in sections through the entire cord, the membrane being left *in situ*.

Peripheral nerves may be hardened in Müller's fluid, care being taken that they do not become brittle by too long soaking in it. The hardening is completed and the specimen preserved in alcohol.

For the hardening of the peripheral nerves, osmic acid is very useful, especially when changes in the myeline are to be sought after.

As osmic acid does not readily penetrate the lamellar sheath so as to come in contact with the nerve fibres, in trunks of any considerable size, the following procedure as suggested by Van Gieson will be found useful: A piece about one-half inch long is cut from the nerve to be examined, and, seizing one end of this segment with a forceps, with another forceps the individual nerve fibres, or small clusters of these, are pulled out of the lamellar sheath and put at once in one-per-cent aqueous solution of osmic acid, in which they remain twenty-four hours, and are then washed and transferred to glycerin, to which twenty-five per cent alcohol is added. In this mixture they may be preserved.

THE THORAX AND ABDOMEN.

To examine these cavities, the body is replaced on its back, and a single straight incision is made from the top of the sternum to the pubes, passing to the left of the umbilicus. For this purpose a large knife should be used, held firmly in the whole hand, and the movement should be mainly from the shoulder. The first incision should divide everything down to the sternum and peritoneum. A short incision should then be made through the peritoneum, just below the ensiform cartilage. Into this opening two fingers of the left hand are introduced and separated from one another, and, the parietes being raised and the sides of the opening being held apart by the fingers, the peritoneum is divided to the pubes, care being taken to hold the knife horizontally so as not to cut the intestines. The skin and muscles are then dissected off from the thorax on both sides as far back as the false ribs.

This dissection should be made by long sweeps of the knife, which should be made to cut with the full blade and not with the point only;

and if the skin and muscles be pulled strongly away from the chest with the left hand, it may be done very rapidly and with a few strokes of the knife. We notice here the amount of subcutaneous fat and the condition of the muscles. In order better to expose the abdominal cavity, the rectus abdominis muscles should be divided transversely beneath the skin just above the pubes, and the abdominal flaps may then be turned freely outward.

General Inspection of the Abdominal Cavity.—We first notice the position and general condition of the viscera. It is best at this stage of the examination to note the condition of the vermiform appendix, and to look over the peritoneal cavity for serum, inflammatory lesions, evidences of perforation, and for the existence of invagination, incarceration, and herniæ of the intestines. A small quantity of reddish serum is frequently found in the abdominal cavity, particularly in warm weather, as the result of commencing decomposition.

It should be remarked here that a variety of striking changes in the character and appearance of the internal organs are produced by putrefaction—changes which are often mistakenly regarded as evidences of disease, and much experience is required in judging correctly of their significance. These changes are, in general, softening and discoloration, both of which may occur as the result of disease. It may be said in general that the post-mortem reddening or hypostases are most marked in the more dependent parts of the organs. Post-mortem softening usually affects entire organs, not being limited to a part, as is often the case in disease. Gray or greenish-brown post-mortem discolorations are apt to appear in those organs or parts of organs which lie in contact with the intestinal canal. Parts of internal organs, such as the liver, which have been the seat of localized congestion during life, may after death take on a dark greenish color.

The *omentum* is usually spread over the surface of the small intestines, but it may be rolled up and displaced in a variety of ways, or may be adherent at some point to the small intestines or the abdominal wall.

The surface of the *small intestines* should be smooth and shining. They may be greatly distended with gas, and thus so completely cover the abdominal viscera that it becomes necessary to let out some of the gas by a small puncture. The transverse colon passes across the abdomen through the upper part of the umbilical region. It may be lower than the umbilicus or higher up against the liver and diaphragm; it may be distended with gas or contracted.

The *liver* is situated in the right hypochondriac and epigastric regions, filling the concavity of the diaphragm. Its upper border reaches, in the linea mammilaris, to the fifth intercostal space; in the linea axillaris, to the seventh intercostal space; close to the vertebral column, to the tenth intercostal space. At the median line the upper border of the liver corresponds to the lower border of the heart. The left lobe extends about three inches to the left of the median line. The lower border of the right lobe usually reaches to the free border of the ribs, while the left lobe is visible for about an inch below the ensiform cartilage. In women, the liver is usually lower than in men.

The position of the liver is affected by changes in the thoracio cavity, forcing it downward; by change in the abdominal cavity, forcing it upward; by constriction of the waist in tight lacing, forcing it either upward or downward; by changes in the size of the organ itself. The liver may not only be displaced downward but dislocated, so that its convex surface faces the abdominal wall, and its posterior edge is turned upward against the diaphragm.

The *stomach* is situated in the left hypochondriac and epigastric regions, extending also into the right hypochondrium; it lies in part against the anterior wall of the abdomen, in part beneath the liver and diaphragm, and above the transverse colon. Its anterior surface, which is directed upward and forward, is in contact above with the diaphragm and the under surface of the liver, and lower down with the abdominal wall opposite to the epigastric region. Its posterior surface is turned downward and backward, and rests on the transverse mesocolon, the pancreas, and the great vessels. To its lesser curvature or upper border are attached the gastro-phrenic ligament and the gastro-hepatic omentum. To the greater curvature or lower border is attached the gastrocolic omentum. Its cardiac orifice communicates with the œsophagus, its pyloric end with the duodenum.

When the stomach is distended, the greater curvature is elevated and carried forward, the anterior surface is turned upward and the posterior surface downward. When distended with food or gas, the organ is prominent; when empty, it may hardly be visible below the ribs; when the intestines are dilated, it may be entirely covered by them.

Before opening the thorax, the hand should be passed up against the under surface of the diaphragm on either side to determine its height. According to Quain, the vault of the diaphragm rises, in the dead body, on the right side to the level of the junction of the fifth rib and sternum, on the left side as high as the sixth rib. Both the relative and the absolute height of the diaphragm vary under a variety of pathological conditions.

If the existence of air or gas in the pleural cavities be suspected, the abdominal cavity should be filled with water and the diaphragm punctured below the level of the fluid. If air be present, it will escape in bubbles through the water.

THE THORAX.

We now leave the abdominal viscera and proceed to the examination of the thorax. With a costatome or a strong knife, the costal cartilages are divided close to the ribs, the clavicles are disarticulated from the sternum, and the latter removed, taking care not to wound the large veins. We first examine the position of the heart and lungs.

The Heart.—The upper border of the heart is on a level with the third costal cartilage; the lower border extends from 1.3 cm. (½ in.) below the lower end of the sternum to the fifth left intercostal space. The left boundary of the heart is situated to the left of the junction of the fifth rib with its costal cartilage, and behind or to the left of a vertical line drawn downward from the left nipple. The right boundary extends about 2.5 cm. (1 in.) to the right of the right edge of the sternum. The portion of the heart uncovered by the lungs is of an irregular quadrangular shape. Its lateral diameter is from 3.8 cm. to 11.1 cm. (1½–4½ inches); its upper boundary varies from the level of the second left costal cartilage to that of the fifth, but it is usually behind the third or fourth cartilage or fourth space.

The area of the heart which is found uncovered will, however, vary much according to the degree to which the lungs collapse after opening the chest. Any disease which diminishes the size of the lungs, or pleuritic adhesions which retract or bind them down, may increase the area of exposed heart. On the other hand, emphysema, pneumonia, or any disease which increases the size of, or retains the air in, the lungs, may diminish the area of exposed heart. The exposed area varies also with the size of the heart itself.

The Pericardium is now opened by a slightly oblique incision on its anterior surface. The existence of serous, fibrinous, or purulent exudation, and of adhesions, is to be noticed. A small quantity of clear serum exists normally in the pericardial sac, and this serum may be blood-stained from beginning decomposition. White thickenings of the pericardium on the surfaces of the heart are often seen; they do not indicate any important disease.

Now that the pericardial sac is open, the position of the heart can be clearly seen. It lies obliquely in the chest, its long axis at an angle of about 60 degrees with that of the thorax. The portion of the heart which is first seen is the anterior surface of the right ventricle; upward and to the right of this is the right auricle, which lies about two-thirds on the right of the sternum and about one-third behind it. Its upper border usually corresponds to the plane of the middle of the anterior end of the second intercostal space on the right side. Its size varies with the amount of blood which it contains. The left auricle lies behind the root of the pulmonary artery, so that only its appendix is visible. The middle of the auricle corresponds to the third costal cartilage. Of the left ventricle only a narrow rim is seen, on the left side of the right ventricle. The pulmonary valve is usually entirely or in part on the left side of the sternum behind the second space or third costal cartilage.

The aortic valve is usually at the level of the third cartilage or the third space, and behind the left two-thirds or half of the sternum. The mitral valve is oblique, the upper end to the left. It is on the level of the third to the fourth cartilage near the middle of the sternum. The tricuspid is oblique, its upper end to the left; the upper end is at the level of the third cartilage, the third space, or the fourth cartilage. The valve is opposite to the middle of the sternum.

The hand should now be passed over the arch of the aorta, to ascertain whether or not an aneurism is present. The heart is then grasped at the apex, raised out of the pericardium, tilted upward and removed unopened by cutting through the vessels at its base.

To determine the sufficiency of the aortic and pulmonary valves, the heart is held horizontally by both auricles, so as not to pull the valves open, and water is poured into the aortic and pulmonary arteries, and we observe how well the valves support the column of liquid. To ascertain the sufficiency of the mitral and tricuspid valves, the auricles are first laid open so as to expose the upper surfaces of the valves. A large pipe is passed through the aorta or pulmonary artery beyond their valves, and a small stream of water allowed to flow into the ventricles. The auriculo-ventricular valves will be swollen upward, and we can observe their degree of sufficiency. The tricuspid valve is normally somewhat insufficient. These water tests, however carefully applied, are not very reliable, since under the most favorable conditions the natural bearings of the valves are not perfectly preserved.

To ascertain the size of the different valvular openings, we introduce the fingers, held flat with their sides in contact, into each of the orifices, and then measure the width of the fingers at the point where they fill the orifice. In this way we find that, under normal conditions in the adult, the aortic orifice measures about 2.5 cm. (1 in.), the mitral valve about 4.5 cm. (1.8 in.), the pulmonary about 3.1 cm. (1.2 in.), the tricuspid about 5 cm. (2 in.).

In order to examine the interior of the heart, we first make an incision through the anterior wall of the right ventricle, close to the septum, and reaching to the apex of the ventricle; through this opening the blade of the enterotome is passed into the pulmonary artery, and the ventricle and artery laid open. With a little care, the incision may be made to pass through one of the points of junction of the valves.

The auricles and ventricles may be empty, or may contain fluid blood, or the so-called heart-clots. These heart-clots are of two kinds—those which are formed some time before death, and those which are formed during the last hours of life and after death. The clots which are formed some time before death are usually associated with organic disease of the heart, especially with dilatation of the ventricles. They are firm, dry, and of whitish color; they may soften or be infiltrated with the salts of lime. They are free in the cavities of the heart, or entangled in the trabeculæ, or firmly adherent to the endocardium. They are composed of coagulated fibrin, or, rarely, of sarcomatous tissue as a complication of sarcoma in other parts of the body. The clots which are formed during the last hours of life and after death are red, yellow, or white. They may be soft or succulent, or quite firm. They may be free in the heart cavities, or be adherent to the trabeculæ, or extend into the large vessels. They are usually most constant and of largest size in the right auricle and ventricle. Such clots may be formed within two hours after death. Clots of this character are a regular post-mortem condition, and of no pathological significance. It is evident, however, if the blood did coagulate in the heart within twenty-four hours before death, that this coagulum could not be distinguished from the ordinary post-mortem clots. If it is supposed, therefore, that a person dies from heart-clot developed a few hours before death, the proof of this must be derived from the clinical symptoms, and not from the autopsy.

The condition of the pulmonary valves and of the endocardium, and the thickness and appearance of the walls of the ventricle, are now noticed; the left ventricle is opened by an incision through its anterior wall, close to the septum, and examined in the same way. We sometimes see the endocardium of the upper part of the left ventricle thick and white without the existence of valvular lesions or any clinical history of disease. The endocardium and valves are often stained red, particularly in warm weather, by imbibition of coloring matter of the blood set free by decomposition. To complete the examination of the cavities, the enterotome is passed into each auricle, carried down into the corresponding ventricle, and an incision made along the outer border of both auricle and ventricle to the apex of the latter. In this way the auriculo-ventricular valves are completely exposed.

After removing the blood, the heart should be finally weighed. In adults, the normal average weight of the heart is about 337 gm. (about 12 oz.). The relative weight of the heart to that of the body is in males about 1 : 158–178; in females, about 1 : 149–176. According to Buhl, the average thickness of the wall of the left ventricle at about the middle of the cavity is from 1.6 cm. to 1.7 cm. (from about $\frac{5}{8}$ to $\frac{2}{3}$ in.); of the right ventricle, from 0.4 to 0.6 cm. (from about $\frac{1}{6}$ to $\frac{1}{4}$ in.).

Generally speaking, the size of the heart corresponds to the size and the development of the individual. In judging of an increase or decrease in its size, we must consider the weight of the organ and the thickness of its walls. If the person die while the heart is contracted, the walls of the ventricles will appear thicker, their cavities smaller than usual. If he die of some exhausting disease, like typhoid fever, or if decomposition have commenced, the heart-walls will usually be flabby and the cavities will appear larger than usual.

Preservation of Specimens.—Parenchymatous and fatty degeneration of the heart-muscle are best studied microscopically by teasing the fresh muscle in one-half-per-cent salt solution, or by examining in the same solution fresh sections made with the freezing microtome. For the study of interstitial changes, fatty infiltration, etc., small pieces of the heart-muscle should be hardened in Müller's fluid. The valves may be stretched on a flat cork with pins, laid for forty-eight hours in a mixture of equal parts of one-sixth-per-cent chromic acid and alcohol, and then transferred to strong alcohol. For the methods of detecting bacteria in ulcerative endocarditis, see section on Bacteria Staining. When the presence of bacteria is suspected, the specimen should be preserved in strong alcohol.

The Pleural Cavities are next examined. The hand is passed into each, and the existence of serous or fibrinous exudations or of old adhesions ascertained. The method of detecting the presence of air has been given above. After the commencement of putrefaction, reddish serum may accumulate in the pleural cavities. This should not be mistaken for the result of disease.

The Lungs.—Each lung is lifted up in turn, the vessels, etc., at its base divided, and the organ removed. If the pleura is very adherent, it is better to strip off the costal pleura with the lung. After inspecting the external surface of the lung, observing its size, shape, color, and consistency, we open the bronchi. For this purpose we use scissors with long, narrow, blunt-pointed blades, one blade a little longer than the other. The lung is held in the left hand with its base upward. We first open the large bronchi which run on the inner side of the lower lobe, afterward those of the upper lobe. Each bronchus should be followed to its smaller ramifications.

We should observe the contents of the bronchi and the appearance of their walls. In the larger and medium-size bronchi, the cartilages in their walls do not form complete rings, but appear shining through the mucous membrane like irregular white patches. This appearance should not be mistaken for a pathological change. In bodies which have been dead for some time, especially in cold weather, the bronchial mucous membrane may be red and swollen as a post-mortem change. The contents of the stomach are sometimes forced, after death, into the pharynx, and thence find their way into the trachea and bronchi, giving them a peculiar reddish and even gangrenous appearance. Bronchitis does not always leave lesions which can be seen after death.

After the examination of the bronchi, the lung is turned over, the vessels, etc., at its root grasped with the left hand, and a long, deep incision made from apex to base. We observe the appearance and texture of the lungs, whether the air vesicles are dilated (emphysematous) or filled with serum, blood, or inflammatory exudation. Fluid can be pressed out of the air vesicles without breaking down the lung tissue. Solid inflammatory exudation, on the other hand, renders the lung more resistant and easily broken down. Attention should be paid to the oozing of purulent or other fluid from the smaller bronchi when the lung is squeezed near the cut surface. It is the rule to find the lower lobes more congested than the upper.

Preservation of the Lungs and Bronchi.—If the lungs have been cut, small pieces from the affected portions of lung tissue or bronchi should be hardened in Müller's fluid, care being taken not to squeeze or handle them unnecessarily. It is better, when the microscopical examination is more important than the macroscopical, not to open the lungs at once, but to fill the air spaces with preservative fluid by means of a funnel attached to a short rubber tube and canula, which is tied into the main bronchus. In this way, not only are the minute structures preserved better, but the air vesicles are filled out and hardened in an approximately natural condition. Care should be taken not to have too great a pressure from the inflowing fluid, since then exudations might be displaced or the lung distorted or ruptured. While the lung is being filled, it should be immersed in a vessel of the same preservative fluid, in which it lies for twenty-four hours. It is then cut into small pieces, and the hardening completed. A variety of hardening agents may be used: Müller's fluid, strong alcohol, or the mixture of equal parts of one-sixth-per-cent solution of chromic acid and alcohol. For general purposes the latter fluid is perhaps the best. If, however, the lung is commencing to decay, strong alcohol will stop the process more quickly, and give as good results as are possible under the circumstances. Alcohol should be used when the lungs are to be examined for bacteria.

It is often desirable, and particularly in cases in which the topography of lesions is to be studied, as in acute miliary tuberculosis, acute and chronic phthisis, infarctions, etc., to inject the blood-vessels with colored gelatin. The lung should, after the injection, be hardened in alcohol.

The Pharynx, Larynx, Œsophagus, and Thyroid Gland.—For the removal of these parts, the incision through the skin should be carried upward as far as practicable—when allowable, to a point one inch below the chin, the head being allowed to hang backward over the edge of the table.

The soft parts are dissected from the larynx, taking care not to cut the thyroid body, and an incision is made through the floor of the mouth, following the internal surface of the inferior maxilla. Through this incision the fingers are introduced into the mouth, the tongue drawn down, the posterior wall of the pharynx divided above the tonsils, and the pharynx and larynx drawn out together. These organs are then pulled downward, and with the aid of the knife the trachea and œsophagus are removed entire, the œsophagus being cut just above the stomach. If the contents of the stomach are to be preserved, as in cases of suspected poisoning, a ligature is put around the œsophagus just below the point at which it is to be cut off.

With the enterotome the pharynx and œsophagus are now slit open upon their posterior surfaces. The mucous membrane thus exposed is examined for evidences of caustic poisons, of inflammation, tumors, strictures, etc. The enterotome is next introduced into the larynx, and this organ and the trachea laid open along the posterior wall. Here we look for œdema of the aryteno-epiglottidean folds (œdema of the glottis), for evidences of catarrhal, croupous, ulcerative, and syphilitic inflammation, and for tumors and lesions of the laryngeal cartilages. Œdema and redness of the larynx may be produced by post-mortem changes, especially in bodies which have been kept for several days in cold weather. A well-marked œdema glottidis during life may leave no trace after death. Putrefactive changes usually commence early in the larynx and trachea.

The thyroid gland is dissected off and examined. Its weight varies a good deal, being, according to Krause, somewhat over 30 gm. (about 1⅛ oz.).

Preservation of the Larynx, Trachea, etc.—The larynx and trachea are freed from superfluous tissue and suspended entire by a thread in a large quantity of a mixture of equal parts of one-sixth-per-cent chromic acid and alcohol. The mixture should be renewed at the end of twenty-four hours, and again on the third and sixth days; at the end of ten days the specimen is washed and transferred to strong alcohol, in which it is preserved. The œsophagus should be stretched on a flat cork with pins, and then treated in the same way. For bacterial study the specimens should be preserved in alcohol. The thyroid may be cut into small pieces, and hardened either in Müller's fluid or alcohol.

THE ABDOMEN.

Returning now to the abdominal cavity, we first dissect off the omentum. If tubercles of the peritoneum exist, they are best seen and studied in the omentum. The colon is then raised and dissected free, to the cæcum on one side and to the rectum on the other. The colon and small intestines are then drawn first to the right and then to the left side, so as to expose in turn the right and left kidneys. As each kidney is brought into view, an incision is made through the peritoneum over the track of the ureter. The ureter is followed through its entire length and its condition ascertained.

The Kidneys are then removed. separating the peritoneum and fat from them with the hand, and dividing the vessels with the knife. The suprarenal capsules, which are attached to the upper end of each kidney, are removed at the same time. The kidneys may be softened by putrefaction, or the surface may have a greenish-gray color, caused by the post-mortem action of putrefactive gases on the hæmoglobin.

An incision is made through the capsule, along the convex border of the kidney, and the membrane stripped off. We notice the degree of adherence of the capsule to the kidney, and also the surface of the latter, whether smooth or roughened, pale, congested, or mottled; an incision is made along the convex surface down to the pelvis, so that the organ is divided into halves. We observe the relative thickness of the cortical and pyramidal portions, as well as the size of the entire organ. To ascertain the latter point, it is well to weigh each kidney; the normal weight is from 130 to 150 gm. (about 4½ to 5 oz.).

It is necessary to remember, however, that in a kidney which is much atrophied there may be an increase of fat in the pelvis, which gives the organ nearly its normal size and weight, while the kidney tissue proper may have in great measure disappeared.

The weight of the kidneys of adults is given by Vierordt in general as about 0.48% of that of the entire body.

We now inspect the kidney tissue more closely, especially the cortical portion. The pyramids consist largely of tubes running in nearly straight lines from the apex to the base of each pyramid. These straight tubes pass from the pyramids into the cortex in bundles, called medullary rays, many of them retaining their straight course until they nearly reach the surface of the kidney. These straight tubules send off branches on all sides of the rays, which become convoluted, form Henle's loops, and finally terminate in the glomeruli or Malpighian bodies. In this way the cortex of the kidney, as seen in section, is divided into alternate bands of straight tubes, convoluted tubes and glomeruli; both sets of bands being perpendicular to the surface of the kidney, and called respectively medullary rays and labyrinths. About the convoluted tubules and glomeruli is a rich venous plexus; and since after death the blood usually remains in this plexus and in the glomeruli, the bands containing the convoluted tubules, *i.e.*, the labyrinths, usually appear red, while the medullary rays are grayish-white. In a normal kidney, therefore, the cortex should be regularly striped in narrow alternating red and whitish bands.

The average thickness of the cortex of the kidney is about one-third of an inch.

If there be extensive congestion, the entire cortex is red. If the epithelium of the tubules degenerates and fills them up, or if there are considerable changes in the interstitial tissue, the regular bands are lost and the cortex is irregularly mottled. If the tubular epithelium becomes filled with fat-globules, this is indicated by an opaque yellow color of the affected parts; in many cases, therefore, the existence of kidney disease can be recognized with the naked eye.

If waxy degeneration be present to a marked extent, it may be manifest by a peculiar translucent appearance of the affected parts, but in most cases it is necessary to apply reagents to demonstrate it satisfactorily. The cut surface of the kidney is washed with water to free it from blood, and repeatedly brushed with an aqueous solution of iodine (iodine 1 part, potassium iodide 3 parts, water 100 parts). The glomeruli and the blood-vessels are most frequently affected, and, if so, they will appear as mahogany-colored dots and lines on a yellow ground.

The pelvis should be examined for inflammatory lesions and calculi. Sometimes a whitish fluid is seen in the pelvis, and can be squeezed from the papillæ; this is produced by a post-mortem desquamation of the epithelium, but is liable to be mistaken for pus.

Preservation of the Kidney.—If the kidney be not opened, the blood-vessels may be injected through the renal artery with alcohol; or, which is better, after washing out the blood-vessels with one-fifth-per-cent solution of chromic acid, the organ may be injected slowly under a low pressure with "*Delafield's* osmic acid mixture" (see p. 44).

After filling the kidney well with this solution, the vessels are tied and the entire organs placed in eighty-per-cent alcohol for twenty-four hours. It is then cut into small pieces and preserved in eighty-per-cent alcohol.

Instead of injecting the organ, small pieces may be cut out and hardened in the osmic acid mixture. This in most cases will be the routine practice, as the osmic acid is expensive and the operation of injection somewhat troublesome.

Kidneys which are to be examined for the presence of bacteria should be cut into small pieces and placed at once in strong alcohol, which should be changed once or twice, and in which they are permanently preserved.

The Suprarenal Capsules are in the fœtus of an ovoidal, in the adult of a triangular shape. They are situated at the upper and inner border of the kidney, to which they are loosely attached by connective tissue. On the anterior surface is an irregular fissure, called the hilus, from which the veins emerge. The size varies considerably, but in the adult the average vertical diameter is from 3.2 cm. (1¼ in.) to 4.5 cm. (1¾ in.); the transverse diameter about 3.2 cm. (1¼ in.), and they are from 4.2 mm. (⅙ in.) to 6.4 mm. (¼ in.) in thickness. They weigh in the adult from about 4 gm. (1 ʒ) to 8 gm. (2 ʒ). They are composed of a cortical and medullary portion, the cortex forming a yellowish shell around the dark-red or brown medulla. They are inclosed in a connective-tissue capsule, from which fibrous processes extend inward, dividing the gland into a series of irregular chambers. Those in the cortex are mostly elongated, giving this portion a striated appearance, while those in the medulla are polyhedral. It is in these spaces that the parenchyma cells lie. The suprarenal capsules readily decompose; the inner layer of the cortex may soften and break down, so that the outer zone forms a sort of cyst filled with reddish-brown broken-down substance. Hypertrophy, tuberculosis and cheesy degeneration, fatty degeneration, and tumors are to be looked for.

Preservation.—The suprarenal capsules should be hardened in Müller's fluid or in strong alcohol.

The Spleen.—This organ has, when removed from the body, the general shape of a flattened ellipsoid, most curved on its external and posterior surface. It is situated in an oblique position on the left side of the stomach, and between its cardiac end and the diaphragm. The vessels are given off from its inner surface, which is crossed by a more or less well-marked vertical ridge. The point of emergence of the vessels is called the hilus. Its long diameter extends from the seventh intercostal space to the eleventh rib. Its upper portion is separated from the ribs by the lungs; its lower portion by the diaphragm.

Its usual length in the adult is, according to Vierordt, from 12 to 13 cm. (about 4¾ to 5 in.); its breadth from 7 to 8 cm. (about 2¾ to 3 in.); its thickness 3 cm. (about 1¼ in.). Its average weight is about 171 gm. (about 7 oz). The dimensions of the spleen as given by Krause are somewhat greater than the above. But its measurement and weight vary considerably within the limits of health. It is in these respects the most variable organ in the body. In old age the average weight gradually diminishes.

The spleen is inclosed in a fibrous capsule covered with peritoneum. The parenchyma is formed of blood-vessels and fibrillar connective tissue, and of a soft, dark-red pulp in which are imbedded whitish spheroidal or elongated bodies, the glomeruli or Malpighian bodies. In the normal human spleen the glomeruli are hardly perceptible to the naked eye, but sometimes they are very plain. Sometimes the fibrous stroma is very apparent; sometimes not.

The size, consistence, and color of the organ vary a good deal without any known cause. Decomposition softens it. Thickenings of the capsule and abnormal adhesions are very common, and often occur without any clinical history indicating disease. We should look for changes in size, pigmentations, hyperplasia of the connective tissue, amyloid degeneration, tubercles, and infarctions.

Not infrequently one or more spheroidal or flattened so-called accessory spleens are found in the vicinity of the spleen; they vary in size from that of a pea to that of a walnut.

Preservation.—In certain diseases of the pulp, leukæmia, leucocythæmia, etc., the tissue should be teased, when fresh, in one-half-per-cent salt solution. For general purposes, small pieces of the organ are hardened in Müller's fluid.

The Intestines.—The rectum is divided, the intestine seized with the left hand, and, being kept stretched, is separated from its attachments by repeated incisions through the mesentery close to the gut, until the duodenum is reached, where it is again cut off. The operation is more cleanly if, before dividing the gut, ligatures are placed around it at either end. The entire length of the gut is now laid open with the enterotome along the mesenteric attachment, the mucous membrane is cleaned with a stream of water and then examined.

In cases of suspected poisoning, a ligature should be placed around the rectal end of the gut and two around the duodenal end, and it is then cut off below the former and between the latter ligatures. The gut is now opened, and the contents emptied into a clean glass jar for delivery to the chemist, care being taken that they be not allowed to touch anything but the inner surface of the jar. After washing the intestine in pure fresh water and examining it, it should be placed entire in another clean jar and sealed.

Cadaveric lividities are very common in the intestines, and are usually most marked in the dependent portions. They are apt to occur in patches, but may be diffuse and very extensive. If the wall of the gut be stretched, they are often seen to be discontinuous, owing to the pressure of the blood from the parts which are squeezed by folds. Small patches of arborescent or diffuse red staining are often seen, formed by the imbibition from the vessels of decomposing hæmoglobin. In the more advanced stages of decomposition, the mucosa may be softened and loosened. A dark purple or brownish discoloration of the entire intestinal wall is frequently seen, either diffuse or in patches. Much experience and careful observation are requisite in forming a correct judgment regarding the significance of changes of color in the intestines. Caution is necessary in distinguishing normal digestive hyperæmia from abnormal congestion. A very considerable congestion may exist without disease. In cholera seasons especially, observers are prone to call the most moderate degree of congestion abnormal.

The lesions ordinarily to be looked for are catarrhal, croupous, and ulcerative inflammations, perforations, hæmorrhages, strictures, tumors, amyloid degeneration, swelling and ulceration of the solitary follicles and Peyer's patches, and pigmentation. For the detection of amyloid degeneration of the mucosa, this structure should be carefully washed and brushed with a solution of iodine (see p. 26).

Preservation.—For the general purposes of microscopic study, portions of the gut should be gently stretched on cork (the mucosa side free) and placed for a few minutes in strong alcohol, and then transferred to eighty-per-cent alcohol, in which the hardening is completed. The transfer to weaker alcohol is to prevent the specimen from becoming brittle.

For obvious reasons, the mucous membrane should be handled as little as possible, for, in the majority of cases, decomposition and softening have already set in at the time of the autopsy, and, under the most favorable conditions, the epithelium is very easily rubbed off.

The Stomach and Duodenum.—We now introduce the enterotome into the duodenum, at its transverse portion, and open it on the convex border. When the pylorus is reached, the incision is carried obliquely over to the greater curvature of the stomach, along which it is extended as far as the œsophageal opening, and the organ examined *in situ;* or, if a more careful examination of the stomach is called for, after ascertaining whether or not the bile-duct is pervious (see below), the duodenum and stomach may be removed together, and the stomach opened and examined on the table. (If poisoning be suspected, a ligature should have been placed earlier in the examination, see above, around the lower end of the œsophagus and the duodenum. The stomach and duodenum are now removed together unopened. They are to be opened in a carefully cleansed glass jar, and after an inspection of the mucous membrane and the contents with the naked eye and a hand-lens, stomach, duodenum, and contents are to be sealed in the jar for the chemist.)

We now look for the orifice of the bile-duct, which will be found about the middle or the descending portion of the duodenum on its concave border. Pressure on the gall-bladder or on the common duct will cause the bile to flow into the intestine if the ducts are pervious. But a sufficient degree of stoppage may exist in the ducts to give rise to marked symptoms of disease without preventing the flow of bile under these conditions, even with a moderate pressure. A long director is now passed into the gall-duct, which is laid completely open; ulcerations, cicatrices, gall-stones, inflammatory lesions, and tumors are looked for. In stricture of the gall-duct, the mucous membrane above will often be found bile-stained, while below it is colorless. At this point, should there be any special reason for doing so, the portal vein, which lies close behind the ductus choledochus, should be opened and examined for periphlebitis, phlebitis, and thrombosis. The mucous membrane of the duodenum and stomach are now rinsed off and examined. Acute inflammations from caustic poisons, chronic catarrhal inflammations, hæmorrhages, ulcers, erosions, swelling of the solitary follicles, and tumors are lesions most frequently seen. We sometimes find a diffuse congestion of the stomach similar to that produced by irritant poisons, as a result of doses of croton oil given just before death.

Preservation.—The same methods should be used as for the intestines (see above). Tumors should be cut into small pieces and hardened in Müller's fluid.

The Liver.—To remove the liver, the diaphragm is first divided on one side of the suspensory ligament as far back as the spine; the suspensory ligament is then divided; then the right and left lobes being in turn raised, the lateral ligaments are severed. Then, seizing the left lobe, the organ is dragged obliquely downward into the abdominal cavity, the remaining attachments being dissected away. The liver is first laid on its superior surface, and the gall-bladder and its contents examined. The character of the gall is to be determined, and gall-stones, inflammatory lesions, and tumors sought for. To determine the actual size of the organ, it should be both measured and weighed. Its size varies greatly in different healthy individuals, but in general it may be said that it measures from 25 to 30 cm. (10 to 12 in.) transversely; from 15.3 to 18 cm. (6 to 7 in.) antero-posteriorly, and about 9 cm. (3½ in.) at its thickest part; the ordinary bulk is about 229 to 252 c.c. (90 to 100 cu. in.); its ordinary weight between 1,550 to 1,860 gm. (50 to 60 oz.).

In children, its weight relative to that of the body is greater than in adults. The liver is increased in size and weight during digestion and by congestion from any cause.

The surface of the liver is now examined, and it is then laid on its lower surface and several deep incisions made from the convex surface downward. The color and consistence of the liver-tissue should be noticed, also the distinctness with which the lobular outlines can be seen; whether or not the centres of the lobules are congested or their peripheries lighter in color than usual; the presence of tumors, tubercles, abscess, ecchinococcus, new connective tissue, and pigmentation. Suspected amyloid degeneration should be tested for by the iodine solution (p. 26).

We often find the surface of the liver of a greenish or very dark-brown color; less frequently the same color extends into the substance of the organ. This discoloration, which is entirely post-mortem, is, like the similar discoloration of other internal organs, produced by the action of the gases or putrefaction on the coloring matter of the blood.

Preservation.—For the study of parenchymatous degeneration, sections of the fresh frozen tissue or small teased fragments should be examined in half-per-cent salt solution. For general purposes, small pieces should be hardened in Müller's fluid. Tumors should be treated in the same way. In many cases of marked cirrhosis, the topography of the lesion is best demonstrated by injecting the organ with blue gelatin through the portal vein and then hardening in strong alcohol.

The Pancreas.—This organ, of a light yellowish-red color, is elongated, irregularly prismatic in shape, and flattened antero posteriorly; the right end, called the head, is broader than the rest, and lies in the concavity of the duodenum. The remainder of the organ, the body and tail, are usually tapering, and lie transversely in the abdominal cavity, the tail reaching to the spleen. Its size and weight vary considerably; its usual length is from 15.3 to 20.3 cm. (6 to 8 in.); its breadth about 3.8 cm. (1½ in.); its thickness about 1.3 to 2.5 cm. (½ to 1 in.); its weight is usually from 70 to 108 gm. (2½ to 3½ oz.) The organ may be rounded instead of flattened; the head and tail may be disproportionately large; the tail may be unusually long or may be divided or curved. The superior mesenteric artery and vein which pass behind the gland are usually partly imbedded in it, but are sometimes completely inclosed.

A longitudinal incision should be made through the whole gland, which may remain *in situ*, and its substance and duct should be searched for calculi, tumors, malformations, and evidences of acute and chronic inflammation and amyloid degeneration of the blood-vessels. The pancreas is frequently of a dark-red color from post-mortem staining.

Preservation.—Portions of this organ should be hardened in strong alcohol, and not in chromic salts, which destroy the gland cells.

THE GENITO-URINARY ORGANS.

The Male Organs.—If the urine is to be examined, it may be drawn off with a catheter; or a vertical incision may be made into the bladder, just above the symphysis pubis, and some of the urine dipped out. The cut end of the rectum should now be grasped with the left hand and raised up, and this and the bladder, prostate gland, etc., dissected away from the pelvis, the knife being carried close to the bone. The bladder is now drawn backward, and the loose tissue close under the symphysis pubis cut. The body of the penis is then shoved backward within the skin and dissected away from behind beneath the symphysis, and finally cut off just behind the glans penis. The penis and bladder are now drawn backward and upward, and the pelvic organs removed together. Or the penis may be removed by sawing away the bones above the pubic arch, and then dissecting away the penis, whose root is thus exposed.

The pelvic organs are then laid on the table, the bladder uppermost; a long director is passed into the urethra, which is opened on its upper surface through its entire length and the bladder widely opened. In the urethra, the presence of strictures, diverticulæ, ulcers, inflammatory lesions is to be noticed; in the bladder, inflammatory lesions, hypertrophies, congestion, and ecchymosis of the mucous membrane and tumors. The organs are now turned over; the rectum opened and examined for varicose veins, hæmorrhages, ulcers, strictures, and tumors. The prostate gland is then cut into and the presence of calculi, inflammatory lesions, hypertrophies, and tumors sought for. Lastly, the vesiculæ seminales are examined, in which, though rarely, we may find evidences of tubercular inflammation and dilatation.

The Testicles may be removed, when necessary, without cutting the scrotum, by enlarging the inguinal canals from within, and crowding the glands through them and cutting them off. The average weight of the adult testicle with its epididymis is, according to Krause, from 15 to 24.5 gm. (about from ½ to ¾ oz.). Inflammatory lesions, tuberculosis, abscesses, and tumors are the most frequent lesions.

Preservation.—The urethral canal and bladder may be pinned open and hardened in alcohol. The prostate, vesiculæ seminales, testicles, and tumors should be hardened in Müller's fluid or alcohol.

The Female Organs.—The position and general condition of the pelvic organs should first be determined by inspection. Abnormal adhesions of the ovaries, broad ligament, Fallopian tubes, and uterus; malpositions of the uterus; subserous tumors of the uterus and ovarian tumors, are frequently observed. Hæmorrhage into the posterior cul-de-sac is sometimes found. The urine should be collected, if necessary, as above directed; the organs should be dissected away laterally, as in the male, care being taken not to injure the ovaries and Fallopian tubes. The bladder is then drawn strongly backward and upward, and dissected away from the symphysis and the pubic arch, and, the point of the knife being carried forward and downward, the vagina is cut off in its lower third, the rectum severed just above the anus, the remaining attachments cut, and the pelvic organs taken out together. If it be necessary to remove the external generative organs, after freeing the lateral surfaces of the internal organs and the bladder, the legs are widely separated and the vulva and anus circumscribed by a deep incision. The tissues close beneath the pubic arch are now dissected away from below and the vulva thrust back beneath the symphysis; it is now seized above the bone, and together with the anus dissected away and removed with the other organs.

The Bladder is first opened and examined. The *vulva* may now be examined for hypertrophies, inflammatory lesions, ulcers, cicatrices, cysts, and tumors. The *Vagina* is opened along the anterior surface; its more common lesions are inflammations, fistulæ, ulcers, tumors, and rarely cysts.

The Uterus.—Before opening this organ, its size and shape should be determined. The adult virgin uterus is a pear-shaped body, flattened antero-posteriorly; the upper portion, or body, is directed upward and forward, whilst the lower portion, the cervix, is directed downward and backward. It is covered anteriorly by peritoneum to a point a little below the level of the os internum; posteriorly, to a point a little below the level of its junction with the vagina. The peritoneal investment separates from the organ at the sides to form the broad ligaments. The uterus is held in position by the broad and round ligaments and by its attachments to the bladder and rectum and vagina. The upper end, the fundus, does not extend above the level of the brim of the pelvis. Its average length is about 7.6 cm. (3 in.); its breadth about 5.1 cm. (2 in.); its thickness about 2.5 cm. (1 in.); its average weight is about 31 to 46 gm. (1 to 1½ oz.). During menstruation the uterus is slightly enlarged, and the mucous membrane of the body becomes thicker, softer, and its vessels engorged with blood; while its inner surface is more or less thickly covered with blood and cell detritus. A description of the complicated changes in the uterus which pregnancy entails may be found in the works on obstetrics. After pregnancy, the uterus does not return to its original size, but remains somewhat larger; the os is wider and frequently fissured.

We not infrequently find in the mucous membrane of the lower part of the cervix, small transparent, spheroidal structures called ovula Nabothi; these are small retention cysts caused by the closure of the orifices of the mucous glands of the part. The more common lesions observed in the uterus are malpositions, malformations, lacerations, ulcerations of the cervix, acute and chronic inflammation of the mucous membrane or muscularis, or both, thrombosis and inflammation of the veins and tumors.

In the infant the uterus is small, the body flattened, the cervix disproportionately large. During childhood the organ increases in size, but the body remains small in proportion to the cervix. At puberty the shape changes and the body becomes larger.

The Ovaries are flattened, ovoidal bodies, situated one on each side and lying nearly horizontally at the back of the broad ligament of the uterus. Their size is variable and they are usually largest in the virgin state. Their average weight is from 3.9 to 6.5 gm. (3 to 5 ℈). They measure about 3.8 cm. (1½ in.) in length, 1.9 cm. (¾ in.) in breadth, and nearly 1.3 cm. (½ in.) in thickness. The sides of the ovary and its posterior border are free; it is attached along the anterior border; to its end is attached the ovarian ligament; to its outer extremity one of the fimbriæ of the Fallopian tube. The ovary is covered on its free surface by cylindrical epithelium, and its surface is less glistening than the general peritoneum. The surface of the ovary is smooth in the young, but becomes rougher and depressed in spots as the process of ovulation goes on. In adult females we usually find corpora lutea in their various stages. We should seek for evidences of acute and chronic inflammations, for tumors and cysts.

The Fallopian Tubes, lying in the upper margin of the broad ligaments, are from 7.6 to 10 cm. (3 to 4 in.) in length. The length often differs considerably on the two sides. They commence at the upper angles of the uterus as small perforated cords, which become larger further outward, and bend backward and downward towards the ovary. They terminate in an expanded fimbriated extremity about 2.5 cm. (1 in.) beyond the ovary. They are covered by peritoneum, and the mucous membrane lining them, continuous with that of the uterus, is thrown into longitudinal folds. Malpositions by adhesions, closure, inflammations, and cysts are the more common lesions. The possibility of tubal pregnancy should be borne in mind.

Preservation.—All of these organs and their tumors may be hardened in Müller's fluid or in alcohol. The vagina should be stretched flat on cork and the cavity of the uterus laid wide open. Great care should be taken not to touch either the internal surface of the uterus or the external surfaces of the ovaries, since in both the epithelium is very easily rubbed off.

It is better, after opening them by a transverse incision, to suspend the ovaries by a thread in a jar of the preservative fluid than to let them lie on the bottom, since the epithelium is thus less liable to be rubbed off.[1]

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[1] Absolute and relative sizes and weights of various parts and organs of the body, and much other valuable statistical data, may be found in *Vierordt's* "Anatomische, Physiologische und Physikalische Daten und Tabellen," Jena, 1888.

AUTOPSIES IN CASES OF SUSPECTED POISONING.

It is always best, in cases of suspected poisoning, to preserve for the chemist not only the stomach and intestine, but the entire liver and brain; or if portions of these only can be saved, these portions should be carefully weighed, as well as the entire organs, and the relative amount of tissue reserved carefully noted at the time It is even well, particularly in cases in which the administration of the readily diffusible poisons, such as arsenic, strychnia, etc., is suspected, to preserve the whole of all of the internal organs, together with a large piece of muscle and bone; since with large quantities of tissue the results of the chemical analysis depend less upon calculations, and are hence more comprehensible to the average jury. In all such cases, jars should, if possible, be procured which have never been used before, and these should be carefully washed and rinsed with distilled water. They should have glass stoppers and be sealed at once and carefully labelled before leaving the hands of the operator. If they can be delivered to the chemist without much delay, they should have no preservative fluid added. If they are to be kept for a considerable time, pending the action of a coroner's jury or for some other reason, a small quantity of pure, strong alcohol may be poured over them. In this case, the operator should be particular to reserve a quantity, at least half a pint, of the specimen of alcohol used, in a clean sealed and labelled bottle, so that this may be tested by the chemist and be proven to be free from the poison. It is better in all cases, however, to avoid, if possible, the use of alcohol. In all autopsies which may have medico-legal importance, full notes should be taken by an assistant as the operation proceeds, carefully read over immediately afterwards, and dated and kept by the operator for future reference. The labelling and disposition of the jars should be recorded in the notes.

EXAMINATION OF THE BODIES OF NEW-BORN CHILDREN.

In examining the bodies of new-born children, we may have to determine, besides the ordinary lesions of disease, the age of the child, whether it was born alive, how long it has been dead, what was the cause of death.

GENERAL INSPECTION.

The Size and Age.—Caspar[1] gives the following description of the fœtus during the different months of intra-uterine life.

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[1] *Caspar*, "Handbook of Forensic Medicine.' Revised German Edition by Liman, 1882, p. 865 *et seq.*; or Sydenham Society Translation.

At the *fourth week*, the embryo is 8 to 13 mm. ($\frac{3}{10}$ to $\frac{5}{10}$ in.) long. The cleft of the mouth and two points indicating the eyes can be recognized in the head. The extremities are represented by little wart-like projections. The heart can be distinguished, the liver is disproportionately large. The umbilical vessels are not yet formed. The entire ovum has about the size of a walnut.

At the *eighth week*, the embryo is 2.3 to 4 cm. ($\frac{9}{10}$ to $1\frac{5}{10}$ in.) long. The head forms more than a third of the entire body; the mouth is very large; the nose and lips can be distinguished, but not the external ear. The hand is longer than the forearm; the fingers are formed, but joined together; the toes look like little buds; the soles of the feet are turned inward. The position of the anus is indicated by a point. The abdomen is closed. All the viscera can be recognized. Centres of ossification are formed in the apophysis of the first cervical vertebra, the humerus, radius, scapula, ribs, and cranial bones. There are rudimentary external genitals, but the sex can hardly be distinguished. The ovum has about the size of a hen's egg.

At the *twelfth week*, the placenta is formed. The embryo is 5 to 6.5 cm. (2 to $2\frac{1}{2}$ in.) long, and weighs about 31 gm. The head is separated from the thorax by a distinct neck. The eyes and mouth are closed. The nails can be perceived on the fingers. The sex can be recognized. The umbilical cord is inserted near the pubes; the muscles begin to be recognizable. The thymus and suprarenal capsules are formed. The cerebrum, cerebellum, medulla, and the cavities of the heart can be recognized. The humerus is 1.7 mm. long; the radius 5.5 mm.; the ulna 6.6 mm.; the femur and tibia 4.4 to 6.6 mm.; the fibula 5.5 mm. The ovum is as large as a goose's egg.

At the *sixteenth week*, the embryo is 13 to 15 cm. (5 to 6 in.) long, and weighs 77 to 93 gm. ($2\frac{1}{2}$ to 3 oz.). The skin is of a rose-red color and has considerable consistency. The formation of fat in the subcutaneous tissue has begun. The scrotum and labia are formed. The face begins to assume its characteristic appearance. There is whitish meconium in the duodenum. The liver is not so disproportionately large, and the gall-bladder is formed; the anus is open. The length of the humerus, radius, and ulna is 1.7 cm.; the femur and tibia 8.8 to 11 cm. The calcaneus begins to ossify at the middle of the fourth month.

At the *twentieth week*, the embryo is 26 to 28 cm. (10 to 11 in.) long; it weighs from 225 to 320 gm. ($7\frac{3}{10}$ to 10 oz.). The nails are quite perceptible. There is a thin down on the head. The head is still disproportionately large, occupying about one-fourth of the body. There is as yet none of the vernix caseosa. The secretion of bile has commenced and stains the meconium. The insertion of the umbilical cord is still further off from the pubes. The liver, heart, and kidneys are large in proportion to the other organs. The convolutions of the brain cannot be recognized. The humerus is 2.8 to 3 cm. long; the radius 2.6 cm.; the ulna 2.8 cm.; the femur, tibia, and fibula, each 2.6 cm. The astragalus and the upper part of the sternum begin to ossify.

From this time on, the length of the fœtus forms an approximately accurate basis for the estimation of its age. *From this period till its maturity, the length of the fœtus, determined in centimetres, corresponds to about one-fifth of the number of months of its age.* From this time on, the weight exhibits marked individual differences, and is therefore a less reliable criterion of its age than is the length.

At the *twenty-fourth week,* the embryo is 31 to 34 cm. (12 to 13 in.) long, and weighs 750 to 875 gm. (24 to 28 oz.). The lanugo and vernix caseosa are formed. The skin is of a dusky cinnabar-red color. The meconium is darker. The scrotum is empty, small, and red; the labia majora are prominent and held apart by the projecting clitoris. The pupillary membrane is present and readily recognized. The length of the humerus and radius is 3.5 cm.; of the ulna, femur, tibia, and fibula, each 3.7 cm.

At the *twenty-eighth week,* the embryo is 36.4 to 39 cm. (14¼ to 15¼ in.) long, and weighs 1,500 to 1,750 gm. (48 to 57 oz.). The hair is more abundant and longer. The great fontanelle measures about 4 cm. (1⅗ in.) in diameter, and all of the fontanelles are readily perceived. The skin is of a dirty reddish color and abundantly beset with the lanugo and vernix caseosa. The large intestine contains much meconium. The humerus is 4.5 to 5 cm. long; the radius 3.7 cm.; the ulna 4 cm.; the femur, tibia, and fibula, each 4.2 to 4.6 cm.

At the *thirty-second week,* the embryo is 39 to 41.5 cm. (15¼ to 16¼ in.) long, and weighs 1,500 to 2,500 gm. (48 to 81 oz.). The skin is lighter in color; the pupillary membrane has disappeared. The testicles are in the scrotum or the inguinal canal; the labia are still widely apart and the clitoris prominent. The nails reach nearly to the ends of the fingers. The humerus is 5 to 5.2 cm. long; the radius 4 to 4.2 cm.; the ulna 4.8 to 5 cm.; the femur 5.2 cm.; the tibia and fibula, each 4.8 to 5 cm. The last sacral vertebra begins to ossify.

At the *thirty-sixth week,* the embryo is 44.2 to 46 cm. (17.4 to 18 in.) long, and weighs about 3,000 gm. (97 oz.). The scrotum begins to become wrinkled and the labia to close. The hair becomes more abundant, while the lanugo begins to diminish in amount.

At the *fortieth week,* the fœtus is fully developed and the term of its intra-uterine life accomplished.

The fresh corpse of a new-born child at term no longer resembles that of the immature fœtus. The skin is firm and pale, like that of an adult. The lanugo has disappeared except on the shoulders. In the majority of cases the hair on the head is 1.5 to 2 cm. (⅗ to ⅘ in.) long. The great fontanelle is, in the average, 2 to 3 cm. ($\frac{8}{10}$ to $1\frac{1}{10}$ in.) long. As determined by an analysis of 661 cases, the average length is 50 cm. (20 in.); the weight 3,256 gm. (105 oz.). The nails are hard and reach to the tips of the fingers, but not to those of the toes. The cartilages of the ears and nose are hard. The labia are more nearly closed. An ossification centre in the lower epiphysis of the femur should be sought for, as its presence is one of the most reliable signs of the maturity of the fœtus. If it is absent, the fœtus is, as a rule, not more than thirty-seven weeks old; but in rare cases it may be absent at term. A centre of ossification 1 mm. (.039 in.) in diameter indicates an age of 37 to 38 weeks, if the child was born dead or died soon after birth. Rarely it is no larger than this at term. A diameter, at birth, of 1.5 to 9 mm. (.058 to .351 in.) indicates an age of 40 weeks. A diameter of less than 9 mm. (.351 in.) indicates, as a rule, that the child has lived some time after its birth; a less diameter than 7 mm. (.273 in.), however, does not prove the contrary.

Twenty-four hours after the birth of the child, the skin is firmer and paler. The umbilical cord is somewhat shrivelled, although still soft and bluish in color. From the *second to the third day,* the skin has a yellowish tinge, and the cuticle sometimes appears cracked. The umbilical cord is brown and dry. From the *third to the fourth day*, the skin is yellower, and the cuticle is apt to separate from the skin. The umbilical cord is of a brownish-red color, flattened, semi-transparent, and twisted. The skin around its insertion is red and congested.

The head should be examined for the marks of injuries. Very commonly some portion of the scalp will be found swollen and infiltrated with blood and serum. This may be the *caput succedaneum* formed during delivery. The mouth and nose should be examined for the presence of any foreign bodies which might have caused suffocation.

The neck should be examined for marks of strangulation. The umbilical cord may be twisted around the child's neck and strangle it. The mark left by the cord is usually continuous, broad, not excoriated, sometimes accompanied by ecchymoses in the skin.

The entire body should be examined for the presence of vernix caseosa, blood, marks of injury, and the existence of putrefaction. It should be remembered that putrefaction is apt to commence earlier in the bodies of young children than in those of adults.

The umbilical cord may be cut or torn. It usually separates by the fifth day, sometimes not until the tenth. If the umbilicus is cicatrized and healed, the child has probably lived for three weeks. A zone of redness around the insertion of the cord may exist previous to birth. Redness and swelling (which may disappear after death) with suppuration can only be found in a child which has lived for several days. The drying and mummification of the cord may take place as well in dead as in living children. It is possible for a child to die by hæmorrhage from a cut or torn cord, either before or after it has breathed.

The extremities may exhibit fracture of the bones. These may occur during intra-uterine life, from injuries to the woman or from unknown causes; or may be produced by violence in delivery, or by injuries after birth.

INTERNAL EXAMINATION.

The Head.—The fontanelles and sutures should first be examined as to their size and for penetrating wounds. An incision should then be made through the scalp across the vertex, and the flaps turned backward and forward, as in the adult. With a small knife, the edges of the bones should be separated from the membranous sutures and the dura mater, beginning low down in the frontal and going back into the lambdoidal suture on either side. The bones are then drawn outward and cut through around the skull, with strong scissors. The brain is removed and examined as in the adult.[1]

Effusions of blood—cephalhæmatoma—may be formed soon after birth, between the pericranium and bone, or, more rarely, between the dura mater and bone. Clots are also found between the dura mater and skull; between the dura and pia mater; more rarely in the substance of the brain, as the result of protracted or instrumental deliveries, or of injuries after birth.

The cranial bones may be malformed, or exhibit the lesions of rickets or caries, or be indented, fissured, or fractured. These latter lesions may be produced during intra-uterine life by injuries to the mother, by unknown causes, by difficult deliveries, or by direct violence after birth.

In cases of chronic internal hydrocephalus in young children in which the ventricles are much dilated and the brain substance thinned over the vertex, the brain is very apt to be torn in removal, and the amount of dilatation thus becomes difficult of determination. It is, therefore, better in such cases to place a pail of water beneath the head, or even immerse the latter in it, and remove the brain in the water. In this way, it floats after removal, supported on all sides. It may now be opened in the water and the extent of the lesion determined at once and parts saved for microscopical examination.

If it be desired to preserve the brain for demonstration of the lesion or for a museum specimen, it should be transferred unopened to a large jar containing a mixture of equal parts of alcohol and water. A portion of the ventricular fluid should now be removed with a syringe provided with a small canula, and replaced by strong alcohol. This may be done by puncturing the ventricles from below. The fluid in the jar, as well as in the ventricles, should be changed in forty-eight hours and then gradually increased in strength until the organ becomes hard. The brain may then be cut transversely across, when the degree of dilatation of the ventricles, etc., will be revealed. The brain, of course, shrinks considerably by this process, but the relative proportions are approximately preserved.

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[1] Or an incision through the bones with a fine saw may be made as in the adult.

The brain is normally much softer and pinker than in the adult, the pia more delicate; both may be much congested or anæmic without known cause. The ventricles contain very little serum. Malformations, apoplexies, hydrocephalus, simple and tubercular inflammatory lesions, are to be looked for.

Spinal Cord.—Extravasations of blood between the membranes of the cord may occur from the same causes as those in the brain. Spina bifida is the most frequent malformation.

The Thorax and Abdomen.—These are opened as in the adult. The *peritoneal cavity* contains a very little clear serum. A red fluid may be produced by decomposition. The peritoneum is often the seat of intra-uterine inflammation.

The Diaphragm.—In still-born infants, its convexity reaches to the fourth or fifth rib. After respiration, it reaches a point between the fourth and seventh ribs. Its position is, however, so variable that it is of little diagnostic importance.

The Thorax.—The *thymus gland*, at this period very large, occupies the upper portion of the anterior mediastinum, covering the trachea and large vessels. Its average weight is about 15.5 gm. (½ oz.). It is usually about 5 cm. (2 in.) long, 3.8 cm. (1½ in.) wide at its lower part. and about .63 to .85 cm. (¼ to ⅓ in.) in thickness. It may be hypertrophied and compress the large vessels, or be inflamed and suppurating.

The *heart* lies more nearly in the median line than in the adult. It weighs from 46 to 108 gm. (1½ to 3½ oz.). The ventricular walls are of nearly equal thickness. The pericardium contains very little serum. A considerable quantity of red fluid may accumulate here as a result of decomposition. There may be small extravasations of blood beneath the pericardium in still-born children and in those born alive. Pericarditis with effusion of serum and fibrin, and endocarditis with consequent changes in the valves, may exist before birth. Malformations and malpositions of the heart cavities and large vessels are not infrequent. The time of closure of the foramen ovale and the ductus arteriosus varies very widely in different cases.

The *pleural cavities* contain very little serum; but decomposition may lead to the accumulation of a considerable quantity of red fluid. Small extravasations of blood in the subpleural tissue may be found in children which have died before birth and after protracted labors. Inflammation, with exudation of serum, fibrin, and pus, may exist before birth.

The *lungs* in a still-born child are small, do not cover the heart, are situated in the upper and posterior portion of the thorax, are of a dark-red color and of firm, liver-like consistence, and do not crepitate. In a child born alive, and which has respired freely, the lungs fill the thoracic cavity, but do not cover the heart as much as in the adult; they are of a light-red or pink color, and crepitate on pressure. If respiration has been incompletely performed, we find various intermediate conditions between the fœtal and inflated states.

If any doubt exists as to respiration having taken place, it is customary to employ the *hydrostatic test.* This is done by placing the lungs, first together, then separately, and afterwards cut into small pieces, in water. It is commonly said that if they sink, the child has not breathed; if they float, it has. This test is not, however, a certain one. Taylor says regarding it:

1. That the hydrostatic test can only show whether a child has or has not breathed, not whether it was born alive or dead.

2. That the lungs of children who have lived after birth may sink in water, owing to their not having received air, or to their being in a diseased condition.

3. That a child may live for some time with the lungs only partly inflated.

4. That a child may live for twenty-four hours when no part of its lungs has been penetrated by air.

5. The sinking of the lungs is no proof that a child has been born dead.

6. That the lungs of children which have not breathed, and have been born dead, may float in water from putrefaction or artificial inflation.

The lesions of inflammation, and vesicular and subpleural emphysema, may be found in the lungs of new-born children.

The *pharynx* should be opened and examined for foreign bodies.

The *larynx* and *trachea* should be examined for the lesions of inflammation and for injuries to the cartilages.

The *thyroid gland* weighs about 12 gm. (ʒ iii.). It may be so enlarged as to interfere with respiration.

The Abdomen.—The *kidneys* are lobulated and proportionately larger than in the adult. There may be ecchymoses on their surface; inflammation; deposits of uric acid and urates in the tubules of the pyramids; cystic dilatation of the tubules, sometimes reaching an enormous size. There may be absence or retarded development of one kidney. Malformations and malpositions of the kidneys are of frequent occurrence.

The *suprarenal capsules* are large. They may be dilated into large cysts filled with blood.

The *spleen* is large and firm. It may be abnormally enlarged, and its surface is sometimes covered with fresh inflammatory exudations.

The *intestines:* In the small intestines, inflammation and swelling and pigmentation of the solitary and agminated follicles are sometimes found. The large intestine usually contains meconium, but this may be evacuated before or during birth. The sigmoid flexure is not as marked as in the adult.

The formation of gas in the stomach and intestines does not usually take place until respiration is established. If decomposition has commenced, however, gas may be formed as a part of the process.

The *liver* is of a dark-red color, is large, and contains much blood. Its size diminishes after respiration is established. The size is so variable, before and after respiration, that it gives little information as to the age of the child. Large extravasations of blood are sometimes found beneath the capsule of the liver, without known cause. A variety of pathological conditions, fatty and waxy degeneration, gummy tumors, etc., may be found.

The *bladder* may be full or empty, both in still-born children and in those which have breathed. Dilatation and hypertrophy may exist during intra-uterine life.

Generative Organs.—The external generative organs in both males and females are more prominent than in adults. The ovaries are high up in the pelvis and large; the cervix uteri is long; the body small and lax, resting forward against the bladder. Phimosis in the male is the normal condition. Malpositions and retarded development of the testicles should be noticed. It should be observed whether the anus is perforate.

The Bones, in suspected cases, should be examined for the lesions of inflammation, rickets, and syphilis.

Preservation.—The various fœtal tissues may be preserved by the same methods as are employed for those of the adult; but as they are very delicate they should be handled with great care, and the preservative fluids changed with sufficient frequency.

GENERAL METHODS OF PRESERVING PATHOLOGICAL SPECIMENS AND PREPARING THEM FOR STUDY.

It is not our purpose in this section to give a complete account of the technical procedures required in the study of pathological specimens, since the methods are for the most part identical with those employed in the study of normal tissues, with which the student or practitioner is presumably familiar before prosecuting pathological studies. We wish simply to give a few brief hints as to the general methods which we have found most useful. Additional details will be found in parts of the book dealing with special tissues and organs.

The Study of Fresh Tissues.—Although for the most part the conditions for the minute study of tissues are more favorable after they have been hardened in some preservative agent, it is yet in many cases very important to examine them in the fresh condition. For this purpose they may be teased apart in a one-half-per-cent solution of sodium chloride and mounted and studied in the same. The same solution may be used for studying semifluid substances, such as exudations from the mucous membranes, pus cells, etc. These preparations are not suitable for permanent mounting, as they do not keep well.

For staining the elements of fresh tissues, particularly the nuclei, and at the same time hardening them so that they can be mounted and preserved for some time, *Carnoy's* solution is of great value. It is made by adding to a saturated aqueous solution of methyl green, 1 per cent of acetic acid and $\frac{1}{10}$ per cent of osmic acid. The tissue is immersed or teased in this solution, and after three to five minutes the color washed off and the specimen mounted in salt solution or in a mixture of equal parts of salt solution and glycerin.

Thin sections of fresh tissues may be prepared by the use of some of the forms of the freezing microtome. That form devised by Thoma and made by Jung, of Heidelberg, is simple, cheap, and effective.

Frozen sections may be stained in an aqueous solution of safranin and then mounted in a mixture of equal parts of glycerin and water.

Decalcifying.—Bones which are the seat of lesions, or calcified tissues, must be freed from lime salts before thin sections can be made from them. This is best accomplished by the use of a saturated aqueous solution of picric acid. The bone or other tissue should be cut into small pieces, not larger than a cubic centimetre, and suspended by a thread in a large quantity of the fluid, which should have an excess of picric acid crystals at the bottom, and should be frequently shaken. Considerable time is required for decalcification by this method, but the results are better than by any other.

If it be necessary to decalcify more rapidly, chromic acid may be used at first and the process completed by nitric acid. The small pieces of bone are suspended at first in a one-sixth-per-cent aqueous solution of chromic acid. After two or three days the strength of the solution is increased to one-quarter-per-cent and after three days to one-half-per-cent. After another week, the fluid should be changed and 1 per cent of nitric acid should be added. This fluid should be renewed every three days until the decalcification is complete, which may be determined by passing a fine needle through the specimen. The specimen should now be thoroughly soaked in water to remove all traces of the acid, and, after lying for a day or two in strong alcohol, is ready for imbedding and section-cutting.

Hardening and Preservation.—In the majority of cases, pathological specimens are best hardened first in *Müller's fluid* and the process completed by alcohol. Müller's fluid is made by the following formula:

Potassium Bichromate........................	2 parts
" Sulphate................	1 "
Water................	100 "

The specimens, which should be cut into small pieces, not more than 1 or 2 cm. square, are placed in a large quantity of the fluid, at least ten times the bulk of the specimen, and allowed to remain for two or three weeks. The fluid should be changed three or four times within the first ten days, and after this as often as the fluid becomes turbid or a sediment forms. After the specimens have acquired considerable consistency, or have been in the fluid for the proper time, they are removed from the fluid and soaked for from twenty-four to forty-eight hours in water, which should be frequently changed. They are then placed in equal parts of alcohol and water for forty-eight hours, and then in strong alcohol, by means of which the hardening is completed. They may be preserved in eighty-per-cent alcohol.

For the special directions for hardening nerve tissues see page 15.

Delafield's Osmic Acid Mixture.—This consists of

One-per-cent solution of Osmic Acid.......... ...	10 cc.
One-fifth-per-cent solution of Chromic Acid... ...	100 "
Ninety-five-per-cent Alcohol	100 "
Acetic Acid	1 "

After remaining for twenty-four hours in this solution, the specimens are transferred to eighty-per-cent alcohol, in which they may be permanently kept. This solution is very efficient in preserving the cells of the parenchyma of such organs as the kidney and liver in a nearly natural condition.

Chromic Acid Mixture.—For many purposes a very excellent hardening may be obtained by using a mixture of one-sixth-per-cent aqueous solution of chromic acid 2 parts, and alcohol 1 part. This fluid, like all others, should be frequently changed, and the hardening may be finally completed with alcohol. This is commonly spoken of simply as the chromic acid mixture.

If specimens are not in a good state of preservation, they will be best preserved by putting them at once into strong alcohol, which may be changed in two or three days.

Although the above is the routine method of hardening tissues, departures from it are occasionally desirable in the preparation of different organs or for the accomplishment of special ends. Thus, in some cases—as in the kidneys, for example—the preservative fluids are brought into more direct and immediate contact with the tissue elements if they are injected under low pressure directly into the blood-vessels. Or by means of a hypodermic syringe the fluids may be thrown directly into the interstices of the tissue by thrusting the needle into them and slowly injecting the preservative agent. This is called *interstitial injection.*

Osmic Acid is of great value for the hardening of small portions of delicate tissues, since it serves to fix the elements in a nearly normal condition and stains them of a brown or black color. It is generally used in one-per cent aqueous solution, the tissues being placed in it when quite fresh and allowed to remain for twenty-four hours. They are now washed in water and may be preserved in a mixture of equal parts of glycerin, alcohol, and water. Such preparations are best adapted for teasing or isolation by other methods than section-cutting. If it is desired to make sections of solid tissues preserved in osmic acid, the latter should be introduced by interstitial injection, and the fragment immersed in the acid for twenty-four hours, and then removed, washed, and placed in strong alcohol. Instead of using the one-per-cent osmic acid pure, very good results are obtained by diluting it with an equal volume each of water and strong alcohol. This is in many cases preferable, since the tissues are not stained so dark by the acid, and are more readily preserved subsequently in alcohol.

Pathological specimens which occur, or are isolated in the form of membranes, should be stretched with pins on a piece of wood or flat cork before being immersed in the preservative fluids.

Minute structures, such as occur in exudations from the mucous membranes and in cyst fluids, renal casts, etc., may be preserved by allowing them to settle, decanting as much of the fluid as possible, and then adding a considerable quantity of Müller's fluid, which after a few days should be decanted and replaced by water. The latter should be renewed by decantation several times, and finally the sediment may be preserved in a mixture of equal parts of glycerin, water, and saturated aqueous solution of picric acid or alcohol.

Imbedding and Section-Cutting.—Some dense tissues, after being well hardened, are sufficiently solid to permit of thin sections being made from them without further preparation, but in most cases very thin sections cannot be prepared without filling the interstices of the tissue with some imbedding material, which gives it greater consistency and holds the tissue elements firmly in their natural relations to one another while the section is being made. Cacao butter, wax, paraffin, and various other substances have been largely used for this purpose, and are very useful; but the more recently employed *celloidin* is by far the most valuable material, and may be used in nearly all cases.

Celloidin, a non-explosive purified form of gun cotton, is best obtained in the form of thin shavings, since it is most easily dissolved in this form. A strong solution is made in equal parts of sulphuric ether and alcohol. The solution should have the consistency of thick molasses. The specimen, having been soaked for twenty-four hours in a mixture of equal parts of alcohol and ether, is placed in the celloidin solution, where it remains until permeated by it. This will ordinarily occur, if the specimen be of moderate size, in from twelve to twenty four hours. For this preliminary soaking the celloidin solution may be a little thinner than above mentioned. If the specimen be small and require but little support, it may now be laid directly on the end of a small cork, and a few drops of celloidin poured around it. In most cases, however, it is better to make a small paper box, in which the specimen is placed in a proper position, and the celloidin poured in around it so as to completely inclose it. In either case, a considerable quantity of celloidin should be poured around the specimen, since the celloidin shrinks considerably in hardening. If sections are to be cut with the microtome, the paper box should be made by winding a strip of filter paper around the end of a straight cork, allowing it to project for a sufficient distance beyond the end. The paper is held in place by tying a thread around it. We have thus a cylindrical box with a cork bottom which projects below it. It is better to use filter paper than sized paper, because the hardening of the celloidin takes place more readily through it.

After the specimen, either free on the end of the cork or in its box, is surrounded by celloidin, it should be allowed to stand for a short time exposed to the air, so that it may harden on the outside by the evaporation of the ether. If the temperature be high, the too rapid evaporation of the ether will cause bubbles to appear in the mass. This should be avoided by covering the specimen with a bell-jar. After the celloidin mass has acquired sufficient hardness on the outside to keep its shape, the whole should be immersed in a mixture of equal parts of alcohol and water, in which the celloidin will harden and acquire a sufficient consistency for cutting in a few hours. When this is accomplished, the paper may be stripped off, and the specimen is ready for section-cutting. A little practice will teach the operator of what consistency to make the celloidin solution, how long to expose to the air, etc.

After the sections have been cut, they may be stained in the usual way (see below), and mounted in glycerin or balsam. If mounted in balsam, the oil of cloves, which is ordinarily used for clearing up the sections, will dissolve the celloidin. For some tissues this does no harm, since they are firm enough to hold together even in thin sections; but in handling friable and delicate tissues it is well to keep the celloidin in place. mounting it with the specimen, with the study of which it does not interfere. This may be accomplished by using the oil of origanum instead of oil of cloves for clearing.

The uncut portion of tissue may be preserved, imbedded in celloidin, by keeping it in eighty-per-cent alcohol.

Section-Cutting may be done by the free hand with a razor ground flat on the lower side, but better sections can be obtained by means of a microtome. One of the most useful of these is Thoma's, which is made of three sizes, the intermediate or the larger one being the more useful. The Schanze microtome is also well adapted for general work, as are some of the American instruments made on the same plan.

Methods of Staining.—Sections of hardened tissues may be stained for microscopical study in a variety of ways, but for routine work the double staining with hæmatoxylin and eosin is most generally useful and applicable to nearly all cases.

The solution of hæmatoxylin is prepared as follows: To make 600 c.c. of the solution, take 400 c.c. saturated solution of ammonia alum, and add to this 4 gm. crystallized hæmatoxylin (Merck's) dissolved in 25 c.c. strong alcohol. This is exposed to the light in an unstoppered bottle for three or four days, when the color will gradually change from a dirty red to a deep bluish-purple color. The solution is now filtered and 100 c.c. each of glycerin and Hasting's wood naphtha are added. After standing for a day or two, the solution is filtered, allowed to stand for another day, and again filtered; and this is repeated until a sediment no longer forms in the fluid.

The solution is now ready for staining, and should be considerably diluted with water as it is used, the best results being obtained by diluting the fluid with from ten to twenty times its bulk of water. The sections are placed in the fluid, and allowed to remain until they have acquired a distinct purple color which persists after rinsing in water. They are now placed for a moment in a dilute alcoholic solution of eosin, and then mounted in glycerin which has been colored lightly with an alcoholic solution of eosin. In this way the nuclei of the cells will be stained of a purple color, while the cell bodies, and to a certain extent the intercellular substance, will be colored a light rose-red.

If specimens are to be mounted in Canada balsam, they are stained with hæmatoxylin as before, and the eosin staining is done by tinging with eosin the alcohol with which the final dehydration of the specimen is accomplished.

Methods of Preserving Specimens for Gross Demonstration and for Museums.—When specimens of diseased tissues or organs are to be preserved entire for exhibition in jars in a museum, it suffices in most cases, after removing superfluous parts and making the requisite dissection, to suspend them in proper position in jars containing thirty-per-cent alcohol. The alcohol should be changed as often as it becomes discolored by blood, and the specimens may be finally preserved in forty-per-cent alcohol. It is desirable that specimens which are to be examined without removal from the jars should be placed in proper position before the alcohol is added, since when they are once hardened it is usually difficult to place them in good positions again; but this may be in most cases deferred for a day or two until a part of the blood is soaked out of them by the first dilute alcohol. The preliminary soaking to remove a part of the blood from specimens may be often advantageously done in strong brine.

Cysts, such as ecchinococcus cysts, small embryos in their membranes, cystic kidneys, etc., may be preserved in a nearly natural condition by placing them in a five-per-cent aqueous solution of chloral hydrate, and after a week replacing this by a ten-per-cent solution of the same, in which they may be permanently preserved. Such specimens are almost equally well preserved in a saturated aqueous solution of chloroform.

Specimens which it is desirable to keep for a few days in a natural condition for the purpose of demonstration may be placed in the following solution:

Water	1,000 c.c.
Common Salt	100 gm.
Saltpetre	25 “
Carbolic Acid	5 “
Glycerin	15 “
Amylic Alcohol	50 “
(Or Ethylic Alcohol	100 “)

This fluid is especially well adapted to the preservation of mucous membranes, such as that of the intestines, in a nearly natural condition for a few days. The specimens should be set in a cool place.

We would most urgently commend to the reader the importance of putting pathological specimens which are to be hardened and subsequently examined microscopically, at the earliest possible moment into the preservative fluids, which should always be abundant. And, furthermore, when specimens are large, it is very desirable to cut them open, so that the fluids may come into direct contact with the tissues. It should be borne in mind that immediately after death or the removal of parts from the body, especially in warm weather, changes commence in the tissues and progress very rapidly, so that in some cases a few hours' or even a few moments' delay will not only render subsequent microscopical examinations difficult and unsatisfactory, but may lead to serious errors. As above stated, Müller's fluid and alcohol are the most generally useful agents. Carbolic acid, glycerin, and usually chloral, should be avoided, and the not uncommon practice of wrapping a specimen in a cloth soaked in alcohol or carbolic acid, and permitting it to remain in this for hours or days, is of no use whatever in preserving specimens of which microscopical examinations are to be made. Almost equally useless is the too common practice of placing a specimen in a bottle which it nearly fills, and pouring a little preservative fluid around it. Not only should the proper fluid be used, but it should be abundant, and the specimen so prepared and arranged that it may come into direct contact with it.

PART II.

CHANGES IN THE CIRCULATION OF THE BLOOD.

CHANGES IN THE COMPOSITION OF THE BLOOD.

DEGENERATIONS.

ANIMAL PARASITES AND BACTERIA.

INFLAMMATION.

TUMORS.

CHANGES IN THE CIRCULATION OF THE BLOOD.

HYPERÆMIA AND ANÆMIA.

There is an important series of changes in the character of the circulation during life which may, when death ensues, either alter considerably in appearance or disappear altogether. Among the more important of these changes are *hyperæmia*—excess of blood in a part; and *anæmia*—deficiency of blood in a part. These conditions and the causes which lead to them will not be described in detail in this book, which has chiefly to do with alterations of the tissue which persist and may be studied after death. Tissues which have been the seat of a temporary, and sometimes of a prolonged, hyperæmia, may show to the naked eye nothing abnormal after death, or they may look redder than normal; they may be œdematous, and more blood than usual may flow from them when incised. On microscopical examination, the blood-vessels may be normal in appearance, or more or less distended with blood. Long-continued hyperæmia may lead to hæmorrhage and transudation, to pigmentation, to a hypertrophy of tissue, or to an atrophy of tissue through pressure, or even to death of tissue.

The paleness which is characteristic of *anæmic* tissues may not be evident after death. Anæmia may lead to no recognizable microscopical changes. On the other hand, if long continued it may induce atrophy and fatty degeneration, and, if excessive, may lead to death of tissue.

HÆMORRHAGE AND TRANSUDATION.

Hæmorrhage is an escape of blood from the heart or vessels. It may occur from a rupture of the walls of the vessels, and is then called hæmorrhage by *rhexis*. The rupture may be occasioned by injury, by some disease of the walls of the vessels which renders them too weak to resist the blood pressure from within, or it may occur from the blood pressure in the thin and incompletely developed walls of new-formed vessels in granulation tissue, tumors, etc.

Under other conditions, without recognizable changes in the walls the vessels, all the elements of the blood may become extravasated by passing, without rupture, through the walls of the vessels. This is called hæmorrhage by *diapedesis*. These hæmorrhages are usually small, but may be very extensive. They usually occur in the smaller veins and capillaries, the cells and fluids of the blood passing out through the cement substance between the endothelial cells. Although no marked morphological changes have as yet been detected which explain this extravasation, it is probable that some change in the nutrition of the walls does occur which renders them more permeable. Hæmorrhage by diapedesis is apt to occur as a result of venous congestion, or when the flow of blood in the smaller vessels has been suspended for some time; or it may result from the action of some poison, or from an injury not leading to rupture; or it may occur in incompletely developed blood-vessels, in tumors and other new-formed tissues.

In the extravasation of blood by diapedesis, the white blood cells may pass through the walls of the vessels, partly at least in virtue of their amœboid movements; the red cells, on the other hand, having no power of spontaneous movement, are, according to Arnold, carried passively through the walls by minute currents of fluid which, under the changed condition, stream in increased force and volume through the endothelial cement substance into the lymph spaces outside.

The altered condition of the blood-vessels leading to hæmorrhage may be local or general, and in the latter case it may either be congenital, as in some cases of the hæmorrhagic diathesis, or it may be the result of some general disease, as scurvy, purpura, etc. The presence of bacteria in the vessels, as in malignant endocarditis and in hæmophilia neonatorum, is believed in some cases to produce changes in the walls of the vessels, leading to extravasation.

Very small hæmorrhages are called *petechiæ*; larger, diffuse accumulations of blood in the interstices of the tissues are commonly called *ecchymoses* or *suggillations*. A complete infiltration of a circumscribed portion of tissue with blood is called a *hæmorrhagic infarction*. A collection of blood in a tumor-like mass is called a *hæmatoma*. Sometimes the elements of the tissue into which the blood escapes are simply crowded apart; sometimes, as in the brain, they are broken down.

The extravasated blood in the tissues usually soon coagulates, although exceptionally it remains fluid for a long time. A certain number of the white blood-cells may wander into adjacent lymph vessels, or they may remain entangled with the red cells in the meshes of the fibrin. The fluid is usually soon absorbed; the fibrin and a portion of the white blood-cells disintegrate and are absorbed. The red blood-cells soon give up their hæmoglobin, which decomposes, and may be carried away or be deposited either in cells or in the intercellular substance at or near the seat of the hæmorrhage, either in the form of yellow or brown granules or as crystals of hæmatoidin. Sometimes all trace of extravasations of blood in the tissues disappears, but frequently their seat is indicated for a long time by a greater or less amount of pigment, or by new-formed connective tissue. Occasionally the blood-mass, in a more or less degenerated condition, becomes incapsulated by connective tissue, forming a cyst.

Transudation is the passage, through the walls of the blood-vessels into the lymph spaces outside, of fluid from the blood, with little or no admixture of its cellular elements. This occurs constantly, to a certain extent, under normal conditions, and forms the commencement of the lymph circulation. But when the amount of fluid passing through the walls of the blood-vessels is increased, or its outflow into the larger lymph trunks is hindered so that it accumulates in undue quantity in the interstices and lymph channels of the tissues, the condition is pathological and is called transudation. An accumulation of transuded fluid in the interstices of the tissues is called œdema; in the serous cavities, dropsy. Its occurrence is usually dependent upon some hindrance to the venous circulation or upon some change in the condition of the blood, which may become more watery or lead to alterations in the walls of the blood-vessels. A simple interference with the outflow of lymph does not usually alone suffice to induce transudation, although it may favor its occurrence. The transuded fluid is usually transparent and colorless or yellowish; it contains the same salts as the blood plasma, but less albumen. It may contain fat, mucin, urea, biliary acids, coloring matter of the bile; fibrinogen is usually present in variable quantity, and rarely fibrin. It may contain endothelial cells from the lymph spaces, and a variable number of red and white blood-cells. The amount of fluid which may accumulate in the tissues varies greatly, depending upon whether they are loose or dense in texture. The fibres and cells of loose tissues may be crowded widely apart; the cells are apt to be more granular than normal, and may be atrophied. Transudations occurring in inflammation usually contain a considerable number of white blood-cells and more or less fibrin, and differ in this from the non-inflammatory transudations; but there is no sharp distinction in some cases between them. The inflammatory transudations are often called *exudations*.

THROMBOSIS AND EMBOLISM.

Thrombosis.—Thrombosis is a coagulation of blood in the heart or vessels during life. The coagulum is called a *thrombus*. Thrombi may lie against the wall of a vessel, only partially filling the lumen, and are then called *parietal thrombi;* or they may entirely fill the vessel, and are then called *obliterating thrombi*.

Thrombi may occur as the result of an injury to the wall of a vessel, or may follow its compression or dilatation; they may result from some alteration of the wall of the vessel by disease or by the retardation of the circulation. So long as the endothelial linings of the vessels are intact, simple retardation of the circulation does not usually alone suffice to induce coagulation; but changes in the endothelium from a great variety of causes, such as inflammation, degeneration, atheroma, calcification, and the presence of tumors and foreign bodies, favor its occurrence.

Thrombi may be composed of fibrin and of red and white blood-cells, intermingled in about the same proportion as in an ordinary extravascular blood-clot. These are called *red thrombi*, and usually occur from some sudden stoppage of the circulation. Other thrombi, usually such as form while the blood is in motion, may consist almost entirely of white blood-cells with a little fibrin, or of these intermingled with blood-plates, or they may consist almost entirely of blood-plates; all of these forms are called *white thrombi*. Red thrombi, when decolorized by changes in the blood-pigment, may somewhat resemble genuine white thrombi. *Mixed thrombi* are usually lamellated, and contain varying proportions of fibrin and red and white blood-cells.

The changes which occur in the thrombus after its formation may be either in the direction of degeneration or organization. In some cases they seem to undergo a simple shrinkage and decolorization. The leucocytes, the fibrin, and the blood-plates may degenerate, forming a granular material which may become infiltrated with salts of lime, forming the so-called *phleboliths*, or *vein-stones ;* in other cases, the thrombi may soften and disintegrate. Certain thrombi contain bacteria or other infectious material, and on softening of the thrombus these may be carried into the circulation, producing very disastrous results. Finally, the thrombus may be replaced by a new formation of vascular connective tissue, itself disappearing as the new tissue is formed. This is called organization of the thrombus, but in reality the new connective tissue is produced, in large measure at least, not from the cells of the thrombus itself, but from the cells of the walls of the affected vessel, from whose vasa vasorum the new blood-vessels of the thrombus also arise. It is possible, however, that the white blood-cells of the thrombus may contribute somewhat to the formation of the new tissue. In this way, the vessel may be completely and permanently occluded, or, more rarely, a channel may be re-established through the new connective-tissue mass.

Thrombi in veins may lead to hyperæmia and œdema; in arteries, to an anæmia whose significance will vary greatly, depending upon the situation of the occluded vessel.

Embolism.—This is the stoppage of a blood-vessel by the arrest in its lumen of some material carried along in the circulating blood. The substance causing the stoppage is called an *embolus*. This may be composed of a great variety of substances. The most common emboli are detached portions of thrombi, and these may have all the variety of structure which thrombi present. Masses of bacteria or other parasites, fragments of the heart valves and of tumors, droplets of fat from the medulla of fractured bones, masses of pigment, bubbles of air, etc., may form emboli. Embolism is, in a majority of cases, confined to the arteries and to the branches of the portal vein.

The primary effect of the stoppage of an arterial trunk is, of course, to largely deprive the region of the body to which its branches are distributed of its normal supply of blood. 'If the branches of the occluded artery form anastomoses with other arteries beyond the point of stoppage, a collateral circulation may be established and the embolus do no harm. If, however, the occluded vessel be a so-called *terminal artery*, that is, one whose branches do not form anastomoses with other arteries, the result of the embolism is quite different. When a terminal artery is occluded by an embolus, the tissue of the affected region usually dies, and there may be an extravasation of blood by diapedesis, leading to the formation of a dark-red, solidified area called a *hæmorrhagic infarction.*[1] The area of infarction corresponds to the region supplied with blood by the occluded vessel, and is usually more or less wedge-shaped.

After a time, the infarction becomes decolorized; inflammatory changes may occur in its periphery, the blood and involved tissues may undergo degeneration and be absorbed, and finally the seat of the infarction may be indicated only by a mass of cicatricial tissue, which frequently contains more or less pigment.

In another class of cases, instead of an extravasation of blood in the affected region, the tissue is simply deprived of nourishment and undergoes necrosis. The affected area is then usually light in color, and is called a *white infarction.* Inflammatory changes may occur in its periphery, and a new connective-tissue capsule form around it, and the dead mass may thus persist for some time, or be gradually absorbed and replaced by cicatricial tissue. The scope of this book does not permit us to consider the somewhat complicated and often obscure reasons why in one case we have hæmorrhagic, in another white infarction, as a result of embolus.

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[1] When an embolus lodges in a terminal artery, and the circulation in the territory supplied by its branches ceases, the pressure from the side of the artery is reduced to zero; but, on the other hand, according to Cohnheim, the venous pressure now makes itself felt in a backward direction, and the capillaries and small veins in the affected region become crowded with blood. This blood is stagnant, however, and the walls of the small vessels, being deprived of their usual nourishment, undergo, it is believed, degenerative changes which favor the occurrence of extensive diapedesis. Thus, in the hæmorrhagic infarction, not only the blood-vessels but the extravascular tissues also are crowded with stagnant blood. The researches of Litten make it seem probable that, in most cases, the back pressure in the region of infarction comes, not from the veins, or not from them alone, but from adjacent arterial twigs which communicate with the capillaries of the affected region.

If the embolic material consists of or contains infectious substances, such as some forms of bacteria, in addition to the mechanical effects of simple emboli, we may have gangrene, suppuration, and formation of abscesses, etc., as the result of the local action of the infectious material, even though this may be present in very small amount.

The organs in which embolic infarctions most frequently occur are the spleen, kidney, brain, lungs; less frequently the retina, liver, and small intestines. Hæmorrhagic infarctions are not liable to occur in the liver from emboli in the branches of the portal vein, on account of the blood-supply which may come to the affected region through the branches of the hepatic artery. On the other hand, embolic abscesses from infectious emboli are of not infrequent occurrence here. Hæmorrhagic infarctions may occur exceptionally in regions not furnished with terminal arteries, as in the small intestines.

CHANGES IN THE COMPOSITION OF THE BLOOD.

Only the more common morphological alterations of the blood will be considered here, and particularly such as may be appreciable after death.

The *coagulability of the blood* and the characters of the resulting clot vary widely, depending partly upon the composition of the blood and partly upon the conditions under which the coagulation occurs. There may be very little coagulation of the blood in death, from the exclusion of air from the lungs, or from diseases and accidents which in any way interfere with the aëration of the blood and permit the accumulation of carbonic acid within it. Thus, in death from strangulation or drowning, many chronic diseases, scurvy, and under many conditions which we do not understand, the blood may remain fluid, or nearly so, after death. On the other hand, in a variety of acute inflammatory diseases, such as rheumatism, pneumonia, etc., very voluminous clots may be formed, although this is by no means constantly the case. The fact that large clots form after death is not conclusive evidence that an undue amount of fibrin-forming elements were present in the blood, nor does the absence of marked coagulation prove a diminution in the blood of fibrin-forming elements.

The blood may be very thick from the removal of its fluid constituents, in diseases associated with excessive serous discharges from the intestines. It is especially marked in some cases of cholera, and is called *anhydræmia.* On the other hand. the blood may contain a large amount of water in proportion to its solid ingredients—albumen and blood-cells. This is called *hydræmia,* and occurs in a variety of diseases of the heart, kidneys, liver, and lungs. In anæmia, etc., the blood looks more or less watery.

There may be a diminution in the number of the red blood-cells and in their hæmoglobin content, as well as changes in their shape. This may be seen in various forms of *anæmia.* The red blood-cells may be unduly pale from a decrease in the amount of hæmoglobin. Varying numbers of spheroidal or irregular-shaped bodies, smaller than the red blood-cells but having the same color, may be found, particularly in marked cases of pernicious anæmia. These are called *microcytes* and may be ill-developed red blood-cells or red blood-cells in a degenerated condition. The number of red blood-cells in extreme cases of anæmia may be diminished to one-tenth of the normal or to about 500,000 in the cubic millimetre. Under these conditions the white blood-cells may remain in normal proportion or they may be increased in number. Fatty degeneration of the heart and blood-vessels, liver, kidney, etc., and capillary hæmorrhages and a change of the yellow into red marrow, are frequent accompaniments of excessive anæmia. This latter change is largely due to the disappearance of the fat. Nucleated red blood-cells are also observed in the red marrow.

Leucocytosis signifies a temporary increase in the number of white blood-cells, and this is usually of moderate amount. It occurs in a variety of acute inflammatory diseases, in profuse suppuration in any part of the body, often in connection with cancerous growths, tuberculosis, etc., and may, as above stated, be associated with anæmia.

Leukæmia (*Leucocythæmia*).—This disease is characterized by a persistent, progressive, and often enormous increase in the number of the white blood-cells. The blood is pale in well-marked cases, but not watery, and the number of white blood-cells may in extreme cases equal or even exceed that of the red. There are sometimes nucleated cells in the blood, of irregular shape and larger than the leucocytes, and others which are smaller. The origin of these cells is not yet certain; the leucocytes are frequently in a condition of fatty degeneration. There may be a decrease in the number of red blood-cells in leucocythæmia. There are marked changes in the liver, spleen, and lymph glands, as well as in the bone marrow, in leukæmia; the degree of involvement of the different organs varying in different cases (see *Leukæmia*, in section devoted to general diseases).

Most of the above-mentioned changes in the morphological characters of the blood are better studied during life than after death, since the redistribution of its elements, owing to the clotting and the effects of gravity, renders the obtaining of a pure sample very difficult or impossible, and the results of examination only in a very general way reliable.

Melanæmia.—In this condition, the blood contains larger and smaller irregular-shaped particles or masses of brown or black pigment. This condition is most frequently the result of intermittent and remittent fever, particularly the severer forms. It may be accompanied by anæmia and leucocytosis. It does not occur in all cases of the above-named affections. It may be transient in character. The pigment may be free, or more usually is inclosed in leucocytes. Under the same conditions, pigment may be deposited in the liver, spleen, lymph glands, bone marrow, and blood-vessels. Owing to the deposit of pigment in the organs, they may assume a gray or slate color. The pigment is supposed to originate in the decomposition of the hæmoglobin.

Pigment which has been taken into the lungs from the air, such as coal-dust, etc., may find its way into the blood either before or after deposition in the bronchial or other lymph glands, and may be afterwards deposited in the spleen and liver.

Foreign Bodies in the Blood.

Various bodies which do not belong there, aside from those above mentioned, may find access to the vessels and mingle with the blood. Pus cells may get into the blood from the opening of an abscess into the vessel, or from some inflammatory change in its walls. Desquamated endothelial cells from the vessel walls, either in a condition of fatty degeneration or in various stages of proliferation, may be mingled with the normal blood elements; also tumor cells of various kinds, fragments of disintegrating thrombi, portions of heart valves, etc. Crystals of bilirubin have been found in the blood in icterus.

Fat, in a moderate amount, is a normal ingredient of the blood during digestion and in lactation. Under pathological conditions it may occur in larger and smaller droplets. *Lipæmia* occurs as a result of deficient oxidation, in diabetes, in drunkards, and in some cases of dyspnœa from various causes. The droplets are small and liable to escape observation.

In many cases of injury, particularly in crushing fractures of the bone, the fat of the marrow finds its way into the blood, and it may collect in large drops in the vessels of the lungs, forming the so-called *fat emboli,* or it may pass the lungs and form emboli in other parts, as the brain, kidneys, etc. Fat embolism in eclampsia is of occasional occurrence.

The fat may be absorbed from the vessels, having produced little or no disturbance; or in some cases it may produce serious results by the stoppage of a large series of vessels in the lungs, brain, or other parts of the body.[1] The fat may be best seen by cutting sections of the fresh tissues with the freezing microtome and staining them at once for twenty-four hours with one-per-cent aqueous solution of osmic acid. They may then be mounted in glycerin.

Air in the Blood, as the result of an opening in the veins, is of occasional occurrence. If the amount of air be small, it appears to be readily absorbed and does little or no harm. If, on the other hand, a large quantity is admitted to the veins at once, it collects in the right side of the heart, from which the contractions of the organs are unable to force it in any considerable quantity, and, the supply of blood being thus cut off from the lungs, death very quickly ensues. It is especially from wounds of the veins of the neck and thorax that the accident is most apt to occur. But it may be due to the introduction of air into the uterine veins in intra-uterine injection, or in the removal of tumors.[1]

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[1] Consult for résumé of this subject, with good bibliography, article by *Park* on "Fat Embolism," New York Med. Jour., Aug. 16th, 1884.

The occurrence of animal and vegetable parasites is considered more in detail in parts of this book devoted to these organisms. It will suffice to mention here that the more important of the animal parasites of the blood are: the *Filaria sanguinis hominis*, the *Distoma hæmatobium*, and the embryos of *trichina* and *echinococcus*, which are of occasional and usually temporary occurrence.

The various species of bacteria which may be found in the blood will be considered in parts of this book in which these organisms are treated in detail.[2]

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[1] Consult *Couty*, "Études exp. sur l'entrée de l'air dans les veines," Paris, 1875, for experiments and older literature; also later article by *Couty*, Arch. de Physiol. nor. et path., 2d ser., T. 4, p. 429, 1877. More recent consideration of the subject in Archiv für klin. Medicin, Bd. 31, p. 441, 1882, by *Jürgensen*.

[2] For literature and detailed consideration of morphological and other alterations of the blood, consult *Cohnheim*, "Vorlesungen über allgemeine Pathologie," Bd. I., 2d ed., or *Orth*, "Lehrbuch der speciellen pathologischen Anatomie," or *Birch-Hirschfeld*, "Lehrbuch der pathologischen Anatomie," 3d ed., Bd. II. For methods of blood examination for clinical purposes, consult *Bizzozero*, "Handbuch der klinischen Mikroscopie," translation into German by Lustig and Bernheimer, or *Jaksch*, "Klin. Diagnose," 2d ed., 1889.

For method of blood counting, apparatus, etc., consult *Freeborn*, "Histological Technique," in Wood's "Handbook of the Medical Sciences."

DEGENERATIVE CHANGES IN THE TISSUES.

Necrosis.—Necrosis is the death of a circumscribed portion of tissue. It may be the result of insufficient nutrition from the cutting off of the blood supply; or it may depend upon the action of destructive chemical agents, extreme degrees of temperature, certain materials produced by the life processes of some forms of bacteria; or it may be due to mechanical injury. The appearances which dead tissues present under the microscope vary greatly. In some cases, we have a simple and gradual disintegration and softening of the tissue, resulting in a mass of degenerated cells and cell detritus, with more or less fluid and various chemical substances resulting from decomposition. The softening of the brain in embolism is an example of simple necrotic softening. In some cases, the dead tissues simply gradually dry and shrivel and become hard and dark colored.

In another class of cases, the dead tissues are permeated by fluids which may be dark red in color from the solution of coloring matter from the blood, and contain bacteria which induce putrefaction with the production of gases and various new chemical substances. The tissues become swollen and granular, and disintegrate ; and finally the whole may form a mass of irregular granules with fat droplets, various forms of crystals, shreds of the more resistant kinds of tissue, and bacteria.

Coagulation Necrosis.—If the supply of blood is shut off from a portion of tissue which is surrounded by, or continuous with, other tissue in which the blood continues to circulate, there results a death and transformation of a peculiar character. The composition of the cells of the tissue is altered so that the cell bodies are shining and translucent, diminished in size, sometimes altered in shape, and the nuclei of the cells disappear. The white infarctions of the spleen and kidneys, the areas of coagulation necrosis in phthisis, and the pseudo-membrane in croupous inflammation of the mucous membranes are the most common examples of this lesion.

If, for example. in the spleen, one of the small arteries is plugged by an embolus, a corresponding portion of the spleen becomes anæmic and appears as a white, wedge-shaped mass, sharply defined from the surrounding splenic tissue. If such a white infarction has existed but a short time, there is hardly any difference between the appearance of its anatomical elements and those of the surrounding spleen, except that they are differently affected by staining fluids. If the infarction is older, the cells are small and shiny and their nuclei cannot be seen.

In croupous inflammations of mucous membranes, the epithelial cells become shiny, the nuclei disappear, and the shape of the cells is changed by the coagulation necrosis so that a number of them together often look like a network of coagulated fibrin.

Cheesy Degeneration.—As commonly used, this term embraces the changes in the tissues which we have just considered under the more appropriate name of coagulation necrosis. But it is also and more properly applied to that form of degeneration in which, under a variety of conditions, the dead tissue elements lose their normal structural features and become converted into an irregularly granular albuminous and fatty material which sometimes tends to disintegrate and soften, sometimes dries and becomes dense and firm, or may become infiltrated with salts of calcium. Thus cheesy degeneration may, and very often does, occur in tissues which are in the condition of coagulation necrosis, but it also occurs in tissues which are not the seat of coagulation necrosis, but which, for a variety of reasons and in a variety of ways, have lost their vitality.

The terms coagulation necrosis and cheesy degeneration, as commonly used, in part actually cover the same degenerative conditions in the tissues. Both are indefinite, and will no doubt remain so until we obtain a more definite knowledge of the lesions which they represent.

Parenchymatous Degeneration (cloudy swelling).—In this condition, the cells of tissues and organs are swollen and filled with small albuminous granules which may be so abundant as to entirely conceal the original cell structure. The granules disappear on treatment with acetic acid, and are insoluble in ether. This degeneration may be present in the parenchyma cells of any inflamed organ, but is most marked and frequent in the liver, kidney, heart-muscle, and mucous membrane of the gastro-intestinal canal. It may occur in infectious or severe febrile diseases, after severe burns, and in poisoning with arsenic, phosphorus, or mineral acids. The cells in a condition of parenchymatous degeneration may regain their normal condition, or become fatty, or disintegrate. In such organs as the liver, kidney, and heart, the gross appearances are often very characteristic; the tissue is swollen and has a less translucent and more dull and grayish look than under normal conditions.

The microscopical study of this lesion is best done in sections of the fresh tissue made with the freezing microtome, or in teased fresh tissue in one-half-per-cent salt solution.

Fatty Degeneration.—This is the conversion of the protoplasm of cells into fat, which accumulates in the cell body. The fat is usually present in the cell in very small particles or droplets, but these may coalesce to form large drops. The protoplasm may even be almost entirely replaced by the fat.

Fatty Infiltration of cells is a common occurrence under normal as well as pathological conditions. The fat is believed to originate outside of the cells and simply accumulate in them, causing a passive atrophy of the protoplasm. In this way fatty infiltration is believed to differ essentially from fatty degeneration, but in many cases a definite distinction between the two is impossible with our present knowledge of the chemistry of cell life. In general, the fat droplets are larger in fatty infiltration than in fatty degeneration, yet to this there are many exceptions. Fat granules and droplets are recognized in cells by their strong refraction, by their solubility in alcohol and ether and their insolubility in acetic acid, and by the black color which they assume when the fresh tissue is treated with osmic acid. Not infrequently feathery clusters of delicate fat crystals occur within the cells. Fatty-degenerated cells may break down and form an oily detritus, in which, especially when much moisture is present, cholesterin crystals may be formed by decomposition of the fat.

To the naked eye, organs in a condition of marked fatty degeneration are usually larger and softer than normal, have a grayish yellow color or are mottled with yellowish streaks or patches, and the normal markings of cut surfaces are more or less obscured.

Fatty degeneration may be due to local or general disturbances of nutrition, from a great variety of causes—disturbances which either directly affect the life processes of the cells themselves, or which produce alterations in their nutritive supply. In addition to its local occurrence, as a result of local disturbances of circulation, in the vicinity of inflammations or in tumors, etc., it is apt to occur in the liver, heart-muscle, and kidney in chronic exhausting diseases and in conditions and diseases to which profound anæmia is incident, or as the result of the action of certain poisons, such as phosphorus and arsenic. Fatty degeneration is, as a rule, a more serious lesion than fatty infiltration.

Tissues in a condition of fatty degeneration or infiltration may be studied by teasing, or cutting the fresh tissue with the freezing microtome and examining unstained, or lightly stained with eosin, in one-half-per-cent salt solution. Or they may be placed, when fresh, for twenty-four hours in one-per-cent solution of osmic acid, and then studied, either after teasing or in sections, in glycerin. A preliminary hardening in Müller's fluid and afterwards in alcohol gives moderately good results if the lesion is extensive. But it should be remembered that in tissues which have been soaked in alcohol the fat is no longer present, its former seat being indicated by clear spaces which are filled with the mounting medium. The fat crystals, however, often persist after prolonged soaking in alcohol.

Amyloid Degeneration (waxy or lardaceous degeneration).—This is a process by which the basement substance of various forms of connective tissue, and especially the walls of the blood-vessels, become swollen and thickened by their conversion into a translucent, firm, glassy, colorless material, albuminous in character. This albuminous material may be present in the tissues in such small amount as to be recognizable only under the microscope, or it may be so abundant as to give a very characteristic appearance to the tissue. Parts in which the lesion is marked are usually larger and contain less blood and feel harder than normal, and have a peculiar dull shining and translucent appearance which varies in character, depending upon the extent and distribution of the degenerated areas and upon its association with other lesions, such as fatty degeneration. It most frequently occurs in the smaller arteries and capillaries, whose lumen becomes encroached upon by the thickening of the walls which the process involves. It is usually the media and intermediary layers of the intima which are earliest and most extensively affected. The change also often occurs in the interstitial connective tissue and membranæ propriæ of organs and in reticular connective tissue. It is both asserted and denied that it may affect the parenchyma cells of organs. We have not been able to find unmistakable evidence of its occurrence in parenchyma cells. These, however, frequently undergo atrophy as the result of pressure from the swollen, degenerated tissue.

It is not yet known whether amyloid degeneration is due to a direct transformation of the tissue, or is an infiltration of the tissue by some abnormal material formed elsewhere and brought to it, or is derived from the blood.

Amyloid degeneration occurs most frequently and abundantly in the liver, spleen, kidneys, intestinal canal, and lymph glands; but it may occur, usually in a less marked degree, in other parts of the body: in the larger blood-vessels, in the interstitial tissue of the heart and mucous membranes of the air passages, and in the generative organs. It may occur locally or appear in various parts of the body at once. It may exist without any known cause, but it most frequently occurs in connection with severe wasting diseases, particularly in those involving chronic suppuration and ulceration, especially of the bones. It frequently occurs in tuberculosis, syphilis, in the cachectic condition induced by malignant tumors, and is occasionally seen in severe malarial infection, dysentery, and leukæmia.

For microscopical examination, the tissue, either fresh or after preservation, should be cut into thin sections, and these deeply stained with one-per-cent aqueous solution of methyl violet; the sections are washed in water and mounted in glycerin. The differentiation between the amyloid and other parts is more distinct if, after staining, the specimen be dipped for an instant in HCl and alcohol 1 : 100, and then carefully rinsed, before mounting in glycerin. The degenerated areas are thus stained rose red, while the normal tissue elements have a bluish-violet color. In some cases, for reasons which we do not know, the amyloid substance does not show a well-marked reaction with methyl violet. Other aniline dyes also differentiate amyloid substance from normal tissues. On treating sections of amyloid tissue with solution of iodine, the degenerated parts acquire a mahogany color. If they are then treated with sulphuric acid, the degenerated portions acquire a greenish or blue color; but the latter reaction is not very reliable.

Corpora Amylacea are small, spheroidal, homogeneous or lamellated bodies which assume a bluish color on treatment with solution of iodine or iodine and sulphuric acid. They are frequently found in the acini of the prostate gland, sometimes in large numbers; in the ependyma of the ventricles of the brain, and in areas of sclerosis of the brain and cord; also in extravasations of blood and in various other situations. They may occur under normal as well as pathological conditions, and are apparently of little importance. They seem to have nothing to do with amyloid degeneration, although they somewhat resemble its products. Some of the tube-casts of the kidney resemble in many respects the corpora amylacea.

Mucoid Degeneration may occur in cells or in intercellular substance. When occurring in cells, it consists, under pathological as under normal conditions, of the transformation of the protoplasm into a translucent, semi-fluid material, occupying more space than the unaltered protoplasm, and hence causing a swelling of the cells. This new-formed material contains mucin in solution, which is precipitated by acetic acid. It occurs under a variety of conditions, sometimes as a morbid increase of a normal function of cells, as in many catarrhs, sometimes as an entirely abnormal transformation. The cells may be entirely destroyed by the accumulation of the mucoid material within them.

In certain cases, as in many tumors, in cartilage, bone, and other tissues, the intercellular substance undergoes conversion into mucin-containing material, losing almost entirely its original structure. The cells in such cases may be affected only secondarily by the pressure which the new-formed material exerts upon them.

Colloid Degeneration is very closely allied, both in chemical and morphological characters, to mucoid degeneration, and in many cases there is no definite microscopical distinction between them. But colloid material is firmer and more consistent than mucous, does not yield a precipitate on addition of acetic acid, and its formation is usually confined to cells; not involving intercellular substance, except by an atrophy which its accumulation sometimes induces. The cells may contain larger and smaller droplets of colloid material, or it may nearly or entirely replace the protoplasm, and accumulate to such an extent as to cause rupture and destruction of the cell. In this way, and by the atrophy of intercellular substance which its accumulation causes, cysts may be formed containing colloid material and cell detritus. Colloid degeneration is of frequent occurrence in certain tumors and in the thyroid gland, and occurs occasionally in other places.

Hyaline Degeneration is the transformation of tissues into a transparent, glassy substance, much resembling amyloid in its morphological characters (Fig. 4); but it does not give the micro-chemical reactions of amyloid, and appears under different conditions. Hyaline substance is quite resistant to the action of acids, and stains readily with acid fuchsin and eosin. It occurs especially in the walls of the smaller blood-vessels in various parts of the body, in voluntary muscle fibres, and is said to sometimes involve interstitial tissue. It has been described as occurring in the brain, lymph glands, and ovaries; in the tubules of the kidney, in the walls of aneurisms, in muscle fibrés, in the lesions of diphtheritis, tuberculosis, and syphilis, in the hyaloid membrane and vessels of the eye, and elsewhere. It is sometimes called vitreous or fibrinous, and also waxy, degeneration. It seems to be, in some ways, allied to coagulation necrosis, but its exact significance and relations to other forms of degeneration, and the conditions of its occurrence, are not yet known.

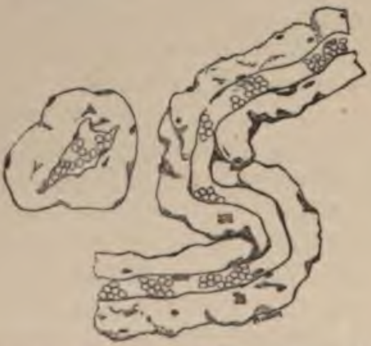

FIG. 4.—HYALINE DEGENERATION OF CAPILLARY BLOOD-VESSELS. From a sarcoma of the optic nerve.

In *Calcareous Degeneration* there is a deposition, either in cells or in the intercellular substance, of larger and smaller granules composed chiefly of phosphate and carbonate of calcium. These particles, when abundant, give hardness, brittleness, and a whitish appearance to the affected tissue. Under the microscope, they appear dark by transmitted, white and glistening by reflected, light. The addition of strong acids causes their solution, usually with the formation of bubbles of carbonic acid gas, whose evolution may be observed under the microscope. Tissues may be nearly completely permeated with the salts, or the latter may be scattered in patches through them. Sometimes large, lamellated concretions are formed in tissues, usually at the seat of some old inflammatory process. Calcification usually occurs in parts of tissues which are dead or are in a condition of reduced vitality, as a result of some antecedent morbid process, usually of an inflammatory nature. Among the most common and important examples of calcareous degenerations may be mentioned those which occur in the valves of the heart and walls of the blood-vessels.

Pigmentation.—The pigment which is present in the body under abnormal conditions may be formed in the body or may be introduced into it from without. It may be deposited in the cells or in the intercellular substance, and is sometimes visible to the naked eye and sometimes not. The pigment occurring in the body may be in the form of yellow, brown, black, or reddish granules, or in crystalline form. In the majority of cases, the pigment is formed by the decomposition of hæmoglobin from extravasated masses of red blood-cells. Parts which have been the seat of long-continued hyperæmia may have a diffuse grayish appearance from the alterations of the hæmoglobin in red blood-cells which have escaped from the vessels by diapedesis. Pigment may be formed in the blood-vessels in severe cases of malarial infection, and circulate in the blood. In another class of cases, various forms of cells seem to be actively concerned in elaborating pigment; this is exemplified in the true melanotic tumors, and the process has its physiological prototype in the formation of pigment in the choroid, skin, and some connective tissues. Pigmentation of tissue from the bile occurs under a variety of conditions, and may be the result of the deposition of granules or crystals. A diffuse staining also frequently occurs from the bile without the formation of solid particles.

In many cases, the mode of formation of pigment is not at all understood. In tissues which are normally somewhat colored, the color may greatly deepen by a simple atrophy of the tissue without the new formation of pigment, as in simple atrophy of the heart-muscle and in atrophied fat.

As examples of pigment introduced into the body from without, we may mention the deposition of minute particles of silver from the internal use of silver nitrate; the coloring of the skin and lymph glands from tattooing; and especially the pigmentation of the lungs and bronchial glands from the inhalation of coal and other dust, which is universally present under the conditions which modern civilization imposes.[1]

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[1] For the literature on the subjects treated of in this chapter, see the extensive bibliography in *Recklinghausen's* "Handbuch der Allgemeinen Pathologie," which constitutes parts 2 and 3 of Billroth and Luecke's "Deutsche Chirurgie," 1883.

PARASITES.

The scope of this work permits us to do little more than enumerate and give a very brief description of some of the more important forms of animal parasites found in man. Among the vegetable parasites, however, the bacteria have assumed such an important place in the consideration of the etiology of certain diseases that they justly claim a somewhat extended notice.

ANIMAL PARASITES.

PROTOZOA.

Among the unicellular organisms, several genera and species occur in the human body, but none are of great pathological significance. A species of *amœba* has been found a few times, in Egypt and elsewhere, in large numbers in the intestinal contents in dysentery. Amœba has also been found in the mouth. The so-called *psorospermiæ*—minute oval structures about .035 mm. long, with a thick capsule and coarsely granular contents—which are of very frequent occurrence in the liver of the rabbit, forming a part of the contents of yellowish, irregular-shaped cysts, have been found three or four times in the liver of man. The organism is more properly called *Coccidium oviforme,* while the spores which it forms are termed psorospermiæ.

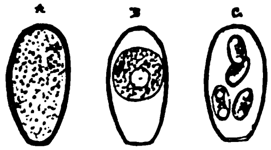

FIG. 5.—COCCIDIUM OVIFORME. From liver of rabbit.

Showing phases in the development of the psorospermiæ, which are seen separate within the capsule in C. After Braun.

Another, smaller form, occurring in the intestinal epithelium of dogs, cats, and rabbits, has been found in two cases in a similar situation in man.

Among the *infusoria* we find in man representatives of several genera. The more common of these are *Cercomonas intestinalis*, a pear-shaped, flagellate structure (Fig. 6) about 0.012 mm. long, making, when alive, rapid movements. It has been found in the evacuations of persons suffering from cholera, typhoid fever, and diarrhœa.

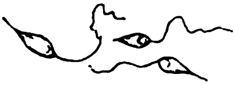

FIG. 6.—CERCOMONAS INTESTINALIS. After Braun.

Trichomonas vaginalis has an oval or pear-shaped body, about 0.01 mm. long, on which are a few cilia, and at the end two or three flagellæ, somewhat longer than the body (Fig. 7). They are of very frequent occurrence in acid vaginal exudations in catarrhal vaginitis. The possibility of mistaking the T. vaginalis for human spermatozoa should be borne in mind in medico-legal examinations, although to an observer familiar with either structure such a mistake could hardly occur.

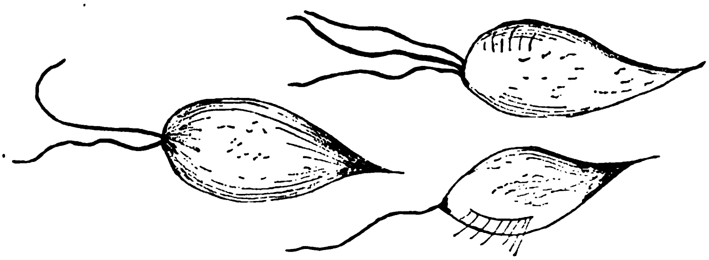

FIG. 7.—TRICHOMONAS VAGINALIS. After Braun.

Some forms of *ciliated infusoria* have been found in large numbers in the intestines in typhoid fever, particularly in the north of Europe. Most of the above-mentioned parasitic protozoa are apparently of little significance and simply accompany, not cause, the lesions with which they are occasionally associated.

WORMS.

TREMATODA (Flukes).—These worms are small, flat, tongue-shaped or leaf-like creatures, with an intestine, and discoidal structures on the under surface, by means of which they attach themselves. There are

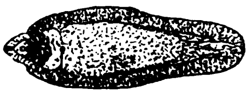

FIG. 8.—DISTOMA HEPATICUM. About natural size.

several genera and species found in man. The most common genus is *Distoma*. Of these, *D. hepaticum* is of most frequent occurrence (Fig. 8).

It is about 30 mm. long, and usually occurs in the gall-ducts and gall-bladder. The embryos are attached generally to water plants, from eating of which the infection is believed to occur. *D. lanceolatum* is more slender, pointed at the ends, 8 to 10 mm. long, and has been found a few times in the gall-bladder. *D. sinense* is a slender worm about 15 mm. long, and has been found in the bile in considerable numbers, particularly in the Chinese. *D. hæmatobium* is a more nearly cylindrical worm; the sexes are distinct; the male from 12 to 14 mm. long, the female 16 to 18 mm. long, and occurs especially in Egyptians, in the portal and other abdominal veins.

CESTODA (Tape Worms).—These important worms consist, in the mature state, of more or less rectangular or elongated segments, each one of which represents a single individual, arranged in a linear series to form a colony, at one end of which, called the head, is a variously formed structure for the attachment of the colony to its host. The neck and head are called the *scolex*, while the segments are called *proglottides*. These worms have neither mouth nor alimentary canal. They are hermaphrodites, the sexes being united in the proglottides. The head and neck (scolex) may exist as an immature form in various tissues and organs where they are incysted, and are often called cysticercus.

Tænia solium is of frequent occurrence in man. It may be several metres in length, and may be coiled up or stretched out in the small intestines. Several worms may be in the gut at one time. The head, about the size of a pin's head (Fig. 9), has a projecting proboscis or rostellum, around which are arranged a double row of horny hooklets. Below these are four sucking discs at the sides of the head. The hooklets of the anterior row are larger than those in the posterior row, and are from 0.16 to 0.18 mm. long. The proglottides, when fully developed, are from 10 to 12 mm. long, and from 5 to 6 mm. wide, but those nearest the head are much shorter and immature. The eggs of T. solium are ovoidal structures, about 0.03 mm. in diameter. The embryo of this worm is most commonly seen in the muscles of the pig as an incysted scolex, commonly called a "measle." It occasionally occurs in man in the muscles, brain, eye, etc., and is called *cysticercus cellulosæ*. It is usually about the size of a pea, but may be as large as a pigeon's egg, and surrounded by a connective-tissue capsule.

Infection with the worm occurs in the human subject from the ingestion of insufficiently cooked "measly" pork, or, in the case of cysticercus cellulosæ, from the ingestion of the eggs, which may, in a variety of ways in uncleanly persons, get into the food.

Tænia mediocanellata (*T. saginata* LEUCKART).—The head of this species is somewhat cuboidal, without either rostellum or hooklets, but with four sucking discs (Fig. 10). The segments are generally broader and shorter than in T. solium, and the worm is usually larger. In the embryonal form the scolex occurs as the *Cysticercus tæniæ mediocanellatæ* in the form of small cysts in the muscles of cattle, from the eating of which in the uncooked condition the infection occurs. This is the most common tape worm in the United States.

Tænia echinococcus.—This worm in the mature condition forms a short, small colony inhabiting the intestine of the dog. The head is about 0.3 mm. in diameter and has a double row of hooklets around the rostellum. The proglottides are three or four in number, the last being the larger. The entire colony is not more than 4 to 5 mm. in length. The significance of this parasite in human pathology depends upon the cysts, called hydatids, which it forms, in the immature or cysticercus stage, in various parts of the body. When the eggs of the mature worm get into the intestinal canal of man, they undergo partial development and find their way into the tissues and organs, most frequently into the liver. Here cysts are formed which become incapsulated by a connective-tissue membrane produced by the inflammatory reaction of the organ.

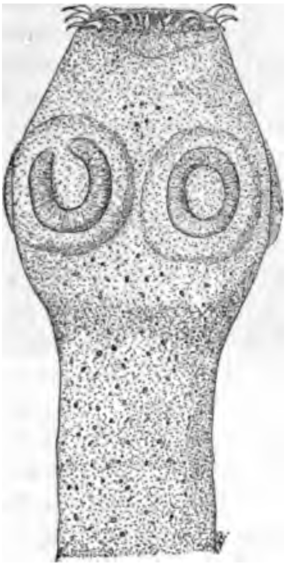

FIG. 9.

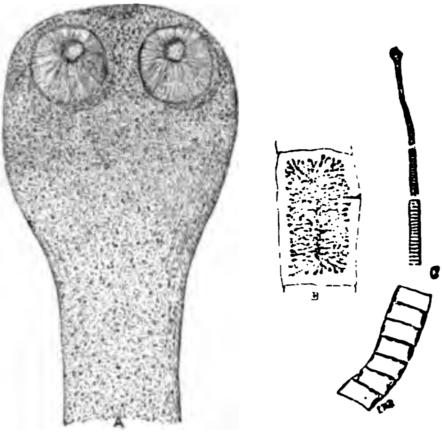

FIG. 10

FIG. 9.—HEAD OF TÆNIA SOLIUM, × about 40.

FIG. 10.—HEAD AND PROGLOTTIDES OF TÆNIA MEDIOCANELLATA

A, Head, × about 15.

B, Mature proglottid, showing generative apparatus.

C, Head and fragments of immature proglottides, showing gradual tapering of the neck. Natural size.

The cyst-wall of the parasite is formed of two layers—an outer, finely lamellated layer called the *cuticula* (Fig. 11), and an inner, granular layer, containing muscle-fibres and blood-vessels, called the *parenchymatous layer*. Inside of the primary cyst, secondary cysts sometimes form, called *daughter cysts*, and within the latter tertiary cysts, called *granddaughter cysts*, may develop. On the inner surface of the cysts, either primary, secondary, or tertiary, the scolices or heads of the immature worm are formed (Fig. 12). These develop in the walls of the pediculated vesicles called *brood capsules*. The walls of these vesicles have a lamellated cuticula and a parenchymatous layer similar to those of the primary cysts. The scolices, of which there may be several in each brood capsule, are similar to the heads of the mature tape-worm. They are about 0.3 mm. in diameter, having a rostellum surrounded by a double row of hooklets and four sucking discs. At the posterior end of the scolex is a pedicle by which it is originally attached to the wall of the brood capsule. Little, lamellated concretions of lime salts are often present in the scolex. The anterior portion of the scolex, the rostellum, hooklets, and suckers, are often invaginated in the posterior portion. The scolices may be free inside of the brood capsules; or, owing to the

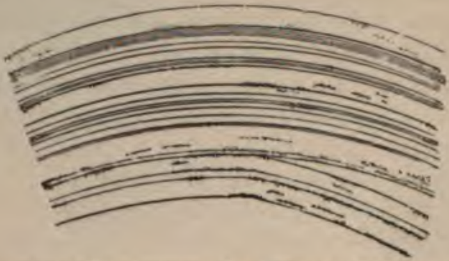

FIG. 11.—CUTICULA OF ECHINOCOCCUS CYST, SHOWING LAMELLATED STRUCTURE.

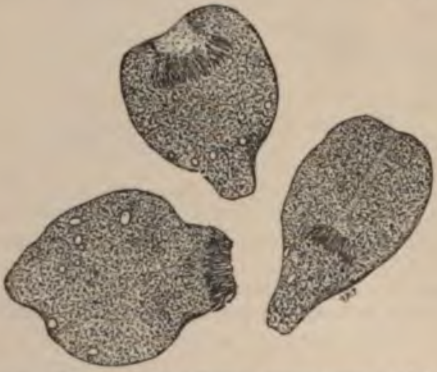

FIG. 12.—SCOLICES OF TÆNIA ECHINOCOCCUS, × 60.
In one the rostellum is projecting, in the others it is withdrawn.

rupture of the latter, they may be free in the cavity of the primary cysts. They may die and degenerate, forming a granular mass in which the hooklets may be imbedded, or the hooklets may be free in the brood capsules or in the primary cysts. Sterile cysts are often found, that is, those in which neither brood capsules nor scolices are developed.

The cysts contain, in addition to the scolices, a clear, gelatinous fluid. This fluid may become turbid by admixture with disintegrated scolices or fragments of the parenchymatous layer, or it may contain fatty detritus, cholesterin crystals, and particles of lime salts. The fluid may be partially absorbed, leaving a thick, grumous material within the cysts, which may become calcified or converted into a stony mass. When the scolices are not found entire, the diagnosis may be made by the discovery of the separate hooklets (Fig. 13), or fragments of the characteristically lamellated cyst-walls. The connective-tissue walls of the primary cysts may become fatty or cheesy or calcified.

Sometimes the secondary vesicles project outward instead of inward, forming a series of cysts outside of the primary one. This variety of development is sometimes seen in man, but is more common in the domestic animals. It is called *Echinococcus scolecipariens* or *exogena*.

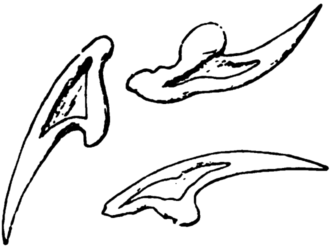

FIG. 13.—HOOKLETS FROM SCOLEX OF TÆNIA ECHINOCOCCUS.

Another variety of echinococcus, called *E. multilocularis*, is almost always found in the liver, and appears to be the result of incomplete and disturbed development of the embryos or cysts. It consists of a congeries of irregular, usually small cysts, surrounded by broad and narrow bands of connective tissue, and sometimes containing gelatinous fluid and scolices or hooklets; but the latter structures are commonly absent or difficult of detection. The whole is often surrounded by a dense connective-tissue capsule which may be calcified. The entire mass often presents an alveolar structure, and was formerly regarded as a tumor—alveolar cancer. The diagnosis may be established by the discovery of the hooklets or scolices, or fragments of the lamellated cuticula.

There are four or five other species of tænia, occurring rarely in man.

T. nana.—This species occurs in the form of small colonies about 15 mm. in length. The rostellum is surrounded by a single row of hooklets. It has been seen once in large numbers by Bilharz in the duodenum of a child which died of meningitis in Cairo.

T. flavopunctata, a species about which little is known, is reported twice in America as occurring in the intestine of young children.

T. madagascariensis, also little known and rare, has been seen in two children in Madagascar.

T. cucumerina.—This species occurs in colonies about 20 cm. long. The head is very small and spheroidal, and has four rows of hooklets. It is frequent in the small intestines of dogs or cats. It occurs occasionally in man. Its scolex inhabits the dog louse, and infection may occur in man by the transference of the lice or the embryos of the parasite to the mouth, as the result of the filthy habit of kissing dogs and cats, or permitting the face to be licked by them.

Bothriocephalus latus.—This, the largest of the human tape-worms, has very broad, quadrangular proglottides. The head is ovoidal and about 2 mm. long and 1 mm. broad. It has no proper sucking discs and no hooklets; but by long grooves on either side of the head the animal attaches itself to its host. The neck is long and filiform. It occurs most frequently in Europe, particularly in the northern provinces. The eggs undergo partial development in water, and are taken up by the pike and eel-pout, and perhaps by other fresh-water fish, from the ingestion of whose flesh in an imperfectly cooked condition the human infection occurs. Two other species of Bothriocephalus have been described as of rare occurrence in man: *B. cordatus* in Greenland and Iceland, and *B. cristatus.*

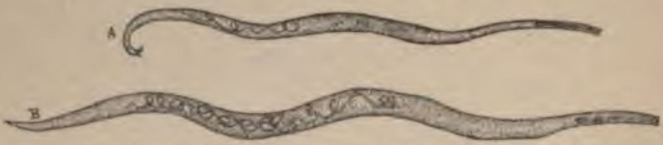

FIG. 14.—ASCARIS LUMBRICOIDES. About half natural size.
A, Male. B, Female. After Perls.

NEMATODA (Round Worms).—These worms are in general cylindrical, elongated, usually pointed at the ends, and sometimes filiform. The surface is sometimes smooth, sometimes irregularly beset with hairs and papillæ, or possesses longitudinal elevated striæ or transverse rings; but the body is not segmented. There is a mouth at the anterior portion, and a ventral anus near the posterior end. The intestine is straight. The sexes are in most forms distinct, the male being in general smaller than the female.

Ascaris lumbricoides.—This is one of the most common of the human intestinal parasites, and is of particularly frequent occurrence in children. It is of a light brownish or reddish color. The female is from 30 to 40 cm. long, and from 5 to 6 mm. thick. The male is somewhat more than half as large (Fig. 14). Both sexes are pointed at the ends, the posterior end of the male being curved into a spined hook. The eggs, from 0.05 to 0.06 mm. in diameter, are surrounded by an albuminous envelope, and are quite resistant to destructive agencies. The mode of development and life history of these parasites are not very well understood. Their usual seat in man is the small intestine, but they may wander into the stomach, and exceptionally get into the mouth, nose, bronchi, gall passages, peritoneal cavity, etc. They may be single in the gut or present in great numbers.

Two other species of ascaris have been found in man. *A. maritima* was found in the vomit of a child in Greenland, in an immature condition. *A. mystax,* a tolerably common form in cats and dogs, has been found a few times in man. It is smaller than A. lumbricoides.

Oxyuris vermicularis (Thread-worm or Pin-worm).—This species is very small; the female has a pointed tail, and is about 1 cm. long. The posterior end of the male, which is about 4 mm. long, is blunt, and after death somewhat curled (Fig. 15). The eggs are produced in great numbers, are oval, and about 0.052 mm. long. This parasite is very common in children, and may be present in large numbers in the colon. They may, in the female, enter the vagina and uterus. This worm is only known to infest the human subject, and infection doubtless occurs by the ingestion of the eggs, which are widely distributed in a variety of ways on many objects, fruits, etc.

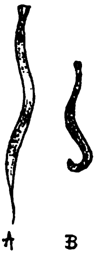

FIG. 15.—OXYURIS VERMICULARIS.
A, Female. B, Male.

Strongylus gigas.—This is a slender red worm, the female being sometimes 1 metre long and over 1 cm. in diameter. It has been found several times in the pelvis of the kidney in man. It is more common in the wolf, fox, horse, seal, and some other animals. *Strongylus longevaginatus.*—The female is about 2.5 cm. long, the male, as usual, shorter. It is of a yellowish-white color, and has been found once in the lung of a boy in Germany.

Dochmius duodenalis.—The female is from 1 to 2 cm. long, the male about 1 cm. long. The body of the male is dilated anteriorly and curved backward. Its mouth is furnished with a chitinous capsule and chitinous claws and teeth. It is found in the small intestine of man in Italy, Switzerland, Egypt, and Brazil. The head is burrowed into the mucous membrane of the host, and the animal is nourished by the blood which it sucks out, and which is usually seen in its intestine. An ecchymosis is produced at the point of attachment, or even severe hæmorrhage, and marked anæmia may be the result of the presence of large numbers of the parasites.

Trichocephalus dispar (Whip-worm).—The males and females are of nearly equal size, 4 to 5 cm. long. A little less than one-half of the body (the posterior portion) is about 1 mm. thick, and in the male is rolled into a flattened spiral, but in the female is but slightly bent. The anterior part of the body is very slender, and is imbedded in the mucous membrane of the host. The eggs are elongated, oval-shaped, about 0.05 mm. long and about one-half as wide, with a thick brown capsule. This parasite is very common in some countries; it dwells in the cæcum in small or large numbers. It is usually of little pathological significance, commonly producing no symptoms. Its developmental history is not well known.

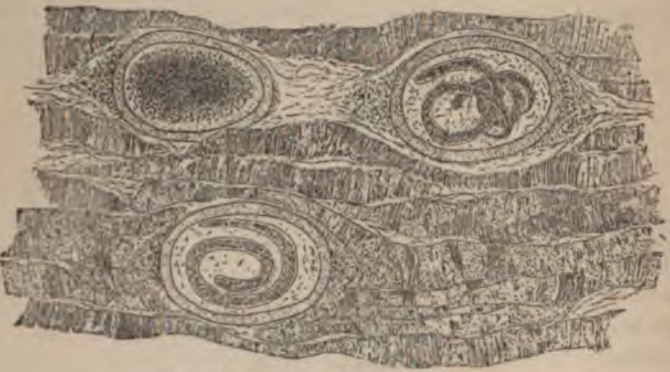

FIG. 16.—TRICHINÆ INCYSTED IN MUSCLE.
In one capsule the worm is dead and its remains calcified.

Trichina spiralis.—The female of this most dangerous and common parasite is, in the mature condition, about 3 mm. long, the male from 1 to 1.5 mm. long; they are filiform in shape and white in color. The young are born in the form of tiny worms about 0.01 mm. in length and somewhat similar to the adult in shape. Infection occurs in man from the ingestion of insufficiently cooked pork. The muscle of the diseased pig contains the embryos of the parasite in an incysted condition. In the stomach, the capsule of the worm is dissolved and the embryos are set free. They very rapidly mature, increasing in size, and the females give birth in the small intestine to very large numbers of young. It is estimated that a single female may give birth to from 1,300 to 1,500 young. These find their way through the mucous membrane and wall of the gut into various parts of the body.

The exact course which they take in getting out of the gut is not fully established; probably they traverse the tissues in different ways. At any rate, they find their way to the voluntary striated muscle tissue, which they penetrate, and enter the muscle fibres. Here they cause a disintegration of the contractile substance, and coil themselves inside of the sarcolemma. In this situation they become incapsulated by material in part furnished by themselves, in part by means of the inflammatory reaction which their presence induces in the connective tissue of the muscle. The worms are surrounded inside the capsule by granular material (Fig. 16). The capsule after a time becomes partially calcified, and in this condition may be readily seen by the naked eye as a tiny white speck. In this incysted state they may remain inactive but living for an indefinite, often for a very long time. Most frequently the cysts contain but one embryo, but they may contain from two to four. The embryo may die and its remains become calcified.

The same course of events transpires when the muscle trichinæ are eaten by the pig or a variety of other animals.

The embryos in the muscle are killed by a temperature of 55° C.

The embryos may mature and a new generation be born within from five to eight days after the ingestion of the diseased meat.

As the result of the presence of these parasites in the body, if the invasion be severe, acute catarrhal enteritis, with diarrhœa and vomiting, high fever, and severe pains. are apt to occur. Œdema of the face and of other parts of the body, broncho-pneumonia, and fatty degeneration of the liver, may be found at the post-mortem examination of cases which have succumbed to the disease. The incapsulated embryos may be found in enormous numbers in various voluntary muscles of the body, but they are most apt to be found, when not very abundant, in the muscles of the neck and larynx, in the intercostals and the diaphragm. They tend to collect towards the tendinous extremities of the muscles. Trichinæ also occur in the rat, cat, mouse, and other animals.[1]

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[1] For the examination of muscles for the detection of the presence of the parasite, small pieces are snipped out with the scissors, and squeezed into a thin sheet between two slides, and examined with a low power. A considerable number of bits of muscle should be examined, particularly from the above-mentioned favorite situations, before excluding them in a suspected case, because they are sometimes present in small numbers. A thorough search is of especial importance in the examination of pork, since, owing to the enormous fertility of the parasites, even a moderate number may give rise to a severe infection.

For the minute examination of the parasite, bits of muscle should be hardened in Müller's fluid and alcohol, and decalcified if necessary, and, after imbedding in celloidin, thin sections cut and stained double with hæmatoxylin and eosin, and mounted

Filaria medinensis (Guinea worm).—This is a thread-like worm; the female, which is alone known, being sometimes as much as 80 cm. long and from 0.5 to 1.7 mm. thick. It is common in the East, and inhabits the subcutaneous connective tissue, in which it often gives rise to abscesses and ulcers. The embryos live for a time free in fresh water, and are then taken up by a species of fresh-water crustacean, in whose body they undergo further development, and by the ingestion of which the infection of the human subject occurs.

Filaria sanguinis hominis.—The embryo of this parasite, which inhabits the blood of men, especially in Brazil, Egypt, and some parts of the Orient, and occasionally occurs in this country, is about 0.35 mm. long, rounded anteriorly, and pointed at the tail (Fig. 17). It has about the diameter of a red blood-cell. It occurs, sometimes in great numbers, in the blood during the night-time, being as a rule absent during the day. It may occur in the urine in connection with chyluria and hæmaturia. The mature female is from 8 to 10 cm. long, and has been found inhabiting the lymph vessels of man, particularly in the scrotum and lower extremities. Owing to the obstructions which it causes in the lymph circulation and to the local irritation which its presence induces, it sometimes gives rise to lymphangiectasis, œdema, abscesses, and perhaps elephantiasis. One of the embryonic stages of development is believed to transpire in the body of a species of nocturnal mosquito. Through the bodies of the dead mosquitoes, which are liable to fall into the drinking-water, it is believed that the spread of the parasite may occur.

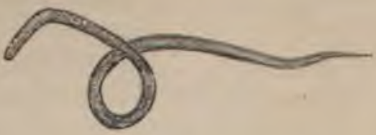

FIG. 17.—FILARIA SANGUINIS HOMINIS.

From a case in the New York Hospital. The specimen was prepared and loaned to the writer by Dr. F. Ferguson.

There are several other species of filaria occasionally found in man which it is not necessary to enumerate here.

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in balsam. Bits of muscle may be also teased, the embryos picked out with a needle, and the cysts either broken open under a lens with the needle, or squeezed under the cover-glass. The embryo worm thus set free may be mounted in a mixture of equal parts of glycerin and picric acid. The adult forms, which may be obtained by feeding rabbits with uncooked trichinous muscle, and examining after the proper interval, may be hardened in Müller's fluid, and mounted in a mixture of equal parts of picric acid and glycerin, or in the same mixture which has been lightly tinged with eosin.

Rhabdonema strongyloides.—A small filiform worm from 1 to 2 mm. in length is found, often in enormous numbers, in the intestines, biliary and pancreatic ducts of man in Cochin China, giving rise to endemic chronic diarrhœa. It has been thought that there are at least two species, which have been described under the generic name *Aguillula*, but recent researches by Leuckart have led him to believe them to be different developmental stages of the same form, for which he suggests the above name.

ARTHROPODS.

The scope of this work does not permit us to enter in detail into the subject of external parasites, which will be found described in treatises on diseases of the skin, or in the general works on parasites referred to below. But, owing to their frequent occurrence and practical importance, we may briefly describe two of the more common forms of arthropods, the "itch insect" and the "louse."

The common "itch insect"—*Sarcoptes hominis* (*Acarus scabiei*)—is shaped somewhat like a turtle, with a chitinous covering, and presents the general appearance seen in Fig. 18. The female is about 0.45 mm. long; the male a little smaller.

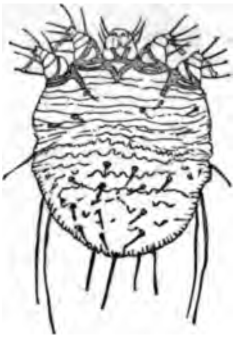

FIG. 18.

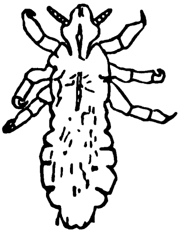

FIG. 19.

FIG. 18.—SARCOPTES HOMINIS—the "itch insect." Female; back view. After Fürstenberg.

FIG. 19.—PEDICULUS CAPITIS—"head louse." Male. After Braun.

The parasite bores little tunnels in the skin in which the eggs are laid and the young hatched. After a few days these bore fresh channels in the skin. For their detection a bit of the superficial layer of the skin is snipped out with curved scissors, dehydrated and cleared up with oil of cloves, and examined under a low power, when the tunnels and the parasites, if present, will be readily visible.

The head louse, *Pediculus capitis*, is from 1 to 2 mm. long, the female being slightly the larger. The general appearance of the insect is seen in Fig. 19.

MODES OF STUDY AND PREPARATION OF THE ANIMAL PARASITES.

The *protozoa* may be studied in the living condition in one-half-per-cent solution of sodium chloride. They may be killed and preserved by allowing a drop of one-per-cent osmic acid to run under the cover-glass, and replacing this after a few hours by glycerin lightly tinged with eosin. The smaller and embryonic forms of the various kinds of parasitic worms may be hardened, best under the cover-glass, with Müller's fluid or osmic acid, and these may be, when the hardening is completed, replaced by dilute, and this by strong alcohol, and the latter finally replaced by eosin-glycerin, in which the specimens are permanently preserved; or they may be stained lightly by tinging the alcohol with eosin, and then cleared up by oil of cloves, and finally mounted in balsam.

It is necessary, however, for detailed study of the larger parasites, to make thin longitudinal and transverse sections from different parts of the body. This can be readily done, even in very small forms, by imbedding the animal—after careful hardening in osmic acid or in Müller's fluid, and afterwards in alcohol—in celloidin, and using the microtome. The sections may be stained double with hæmatoxylin and eosin, and mounted in balsam.

The general arrangement of the generative organs in the proglottides of tape worms may be well seen by staining in eosin or eosin-glycerin after moderate hardening in dilute alcohol, and then squeezing the segment between two glass slides. The itch insect and louse may be soaked for a few hours in turpentine and mounted in balsam.[1]

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[1] *Bibliography.*—Especially to be recommended for detailed descriptions of human parasites, together with practical suggestions for their study, is the small work of *Braun*, "Die thierischen Parasiten des Menschen," 1883, which contains also the more important bibliography.

The more extended classical works of *Cobbold*, "Entozoa of Men and Animals," 1879, and *Küchenmeister* and *Zürn*, "Die Parasiten des Menschen," 2d ed., and the work of *Leuckart*, "Die Menschlichen Parasiten," should be consulted, and contain valuable bibliography. Various forms of external parasites of men and animals are fully described and illustrated in the work of *Mégnin*, "Les Parasites et les Maladies parasitaires chez l'homme," etc., 1880. The plates of *Stein*, illustrating the *Cestoda*, 1882, are carefully executed. In the "Report on Trichinæ and Trichinosis," in 1880, by *Glazier*, Surgeon in the Marine Hospital Service, will be found an illustrated account of the natural history of this parasite, history of the disease, etc., and a section on its occurrence in the United States.

VEGETABLE PARASITES.

PARASITIC FUNGI.

These lowly vegetable organisms, which are of not infrequent occurrence on and in the human body, both in health and disease, embrace the structures commonly known as *moulds* and *yeasts*. Of these, the moulds are of the greatest importance, and these present varying degrees of significance. The scope of this work does not permit us to describe either the natural history of the group or the different forms. For this we refer to works on skin disease and on vegetable parasites.

We may simply mention as the more common and important the *Achorion Schönleinii*, or favus fungus; the *Trichophyton tonsurans*, or ringworm fungus; the *Microsporon furfur*, or the fungus of pityriasis versicolor. A form of *Aspergillus* is of occasional occurrence in the external meatus of the ear and on the membrana tympani. The peculiar white pellicle often seen on the mucous membrane of the mouth and pharynx, sometimes in adults who are the victims of exhausting diseases, but more often in young children (*thrush*), is associated with the fungus *Oidium albicans* (*Saccharomyces albicans*).

Actinomyces (ray fungus).—This fungus grows in radiate masses, especially in the jaws of cattle, but is of occasional occurrence in man. The disease which it causes is called actinomycosis. The fungous mass may form a large tumor in the jaw, by its own growth and by the formation of granulation tissue, which is apt to slough and spread so that not only may the tissues of the tongue, pharynx, larynx, etc., be involved, but nodules of similar character may form in the gastro-intestinal canal, lungs, skin, etc. These nodules were formerly considered to be some form of sarcoma. The fungus forms little yellow masses as large as a millet seed or smaller, which are scattered through the new-formed granulation tissue or mingled with the pus, giving the growths a very characteristic appearance. It is the peculiar radiate grouping of the filaments of the growth (Fig. 20) which gave rise to the name "ray fungus." The disease is propagated from one animal to another by inoculation or by contact of the growth with a wound or an abrasion of the mucous membrane.[1]

For convenience, the actinomyces is here mentioned in connection with the fungi, but its life history and affinities are too little known as yet to enable us definitely to classify it.

The tissue containing actinomyces may be teased in a fresh state and stained in an alcoholic solution of eosin. Sections of the diseased tissue may be stained double, first for an hour with orseille and then with gentian violet

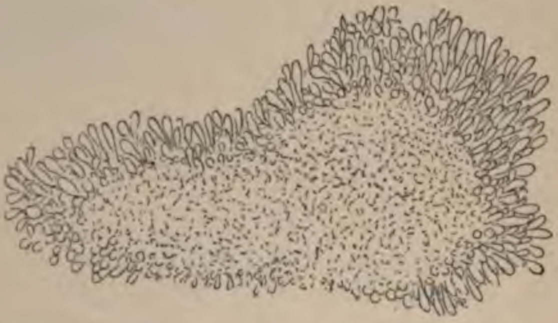

FIG. 20.—ACTINOMYCES BOVIS, showing one of the yellowish masses of the parasite separated from the surrounding tissue.

BACTERIA.

Morphology and Physiology.—Bacteria are minute vegetable organisms of the lowest and simplest form allied to the algæ. They are widely distributed in the air, water, and surface soil, and are particularly abundant among the habitations of men, or wherever animal and vegetable substances are undergoing decay. Putrefaction and putrefactive products are always dependent upon their presence and activity.

The bacteria play a very important rôle in nature in virtue of their power of feeding upon and pulling to pieces dead organic materials. A small part of the new chemical compounds which are thus formed are used by the bacteria themselves for the purposes of their own nutrition and growth, while the rest are set free to serve, sooner or later, as food for other forms of plants or animals. Some of the chemical compounds set free by the growing bacteria are bad-smelling or aromatic gases; some are inert and harmless substances; some are powerful poisons, and may, when they have accumulated in the fluids where they grow, destroy the bacteria which have produced them.

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[1] For general consideration of the fungi and their relations to disease, consult *Flügge*, "Mikro-organismen," 1886.

Bacteria of one kind or another are invariably present in greater or less numbers in the mouth, nose, lungs, and gastro-intestinal canal of men and animals. The largest of them are too small to be seen with the naked eye, and the smallest require for their recognition the most perfect and powerful of our microscopic aids. They have various shapes: spheroidal, rod-like, filiform, or spiral. They are simple cells, multiplying, when the conditions are favorable, with extraordinary rapidity, by transverse division, or by the formation of spores which develop into the adult forms. They are formed of a membrane inclosing the protoplasm, which may appear homogeneous or granular.

They lie sometimes singly, sometimes in pairs or chains, and sometimes in masses imbedded in a transparent gelatinous substance, and are then called *zoöglœa colonies.* Some are capable of performing rapid movements, others not, and the same form may be at one time mobile and at another immobile. Some of them have a cilium at the ends by means of whose vibrations they may perform active progressive movements. Some are surrounded by a homogeneous envelope called a capsule.

They require for their nourishment nitrogen and the hydrocarbons; oxygen is necessary for the proliferation and activities of some forms, and for others not.

They are active only in the presence of moisture, but when this and other conditions favoring their activities fail they do not necessarily die, but some forms may remain, either as spores or as fully developed organisms, for long periods wholly dry and inert, but capable of resuming their activity whenever they are again restored to favorable conditions.

Some are and some are not very sensitive to changes of temperature. At a temperature below + 5° C. they are incapable of activity or proliferation, but some forms are not killed by a temperature of −111° C. As the temperature is raised, their activities increase up to a certain point. It may be said in general that they are most active at about the temperature of the body, although species differ considerably in this respect. In fluids, many bacteria are killed by a temperature of 80° C., and all are killed by a temperature of 100° C. (the boiling point of water) if this be long enough continued. When dry, they resist much higher temperatures than when moist. The spores are, as a rule, more resistant to high temperatures than the bacteria themselves; some having been exposed, dry, to a temperature of 140° C. without destruction of life. Fluids containing the spores of bacteria which resist very high temperatures may be sterilized by boiling for a short time, then being allowed to stand at ordinary temperatures for several hours, and then again boiling; this process being repeated several times. In this way, although the spores themselves are not killed by the heat, the bacteria into which they develop during the intervals are killed, so that finally the medium is entirely freed from both living spores and adult bacteria.

Certain disinfecting agents, when brought into contact with bacteria, are capable of greatly reducing their activities, or destroying them altogether; but different forms differ greatly in their power of resisting the action of these agents. The spores of certain bacteria are exceedingly resistant, much more so than the bacteria themselves, to the action of disinfecting agents. Among these disinfectants may be mentioned sulphurous acid. bromine, creolin, carbolic acid, and especially solutions of corrosive sublimate, which is very inimical to the life of most bacteria and their spores, even in extremely dilute solutions.

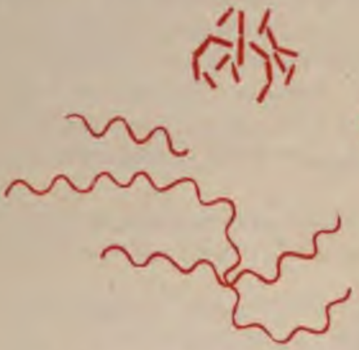

FIG. 21.—DRAWING OF THREE TYPICAL FORMS OF BACTERIA ILLUSTRATING THE THREE CLASSES. Stained with fuchsin.

Classification of Bacteria —The systematic study of these organisms by exact and reliable methods is of such recent date, they are so small and difficult to examine completely, and so much uncertainty still exists about the character and life history of many known forms, that a satisfactory and scientific classification of them is not yet possible. For the sake of convenience, however, we group the various known species according to their shape :

I. *Spheroidal bacteria.*
II. *Rod-shaped or filiform bacteria.*
III. *Spiral-shaped bacteria.*[1]

This classification or grouping of the bacteria is temporary and provisional, and as our knowledge increases will, no doubt, be essentially modified or altogether replaced ; but it is at present almost indispensable for purposes of study.

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[1] In addition to these, there are a few forms which present such variable shapes in different phases of their growth that they form a rather indefinite class by themselves.

When the bacteria are growing under unfavorable conditions, or when they are old or dead, they may assume various distorted, bizarre shapes, which are called *involution forms.*

I. SPHEROIDAL BACTERIA.

The most common and important genus of this group of bacteria is *Micrococcus*, of which there are many species. Some of these are capable of producing coloring materials of various kinds in the nutrient media; others induce different forms of fermentation; still others occurring in the body in connection with various diseases are called *pathogenic micrococci*. The cells of most of the species of micrococci are very small, having, for the most part, a diameter of less than 1 μ (0.001 mm.); but some forms are considerably larger. They occur either singly or joined together in pairs—*diplococcus;* in longer or shorter beaded chains—*streptococci;* or in zoöglœa masses (Fig. 22). They are believed never to exhibit spontaneous movements, although liable to show the Brownian movement. The species differ from one another in size, sometimes slightly in shape, in mode of grouping, in refractile power, and in their physiological activities.

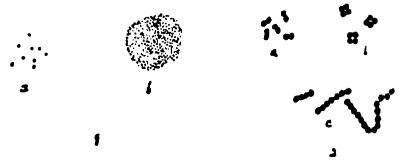

FIG. 22.—SPHEROIDAL BACTERIA (GENUS MICROCOCCUS).

1. Micrococcus from ulcerative endocarditis. *a*, single; *b*, zoöglœa.

2. Micrococci cultivated from laboratory dust. *a*, in pairs (diplococcus form); *b*, in tetrads, M. tetragenous; *c*, in chains (streptococcus form). Stained with fuchsin.

Among the color-producing micrococci may be mentioned the *Micrococcus luteus*, which is common in the dust of the air, and, when growing in masses, forms yellow droplets on the surface of boiled potatoes or other nutrient media.

The *Micrococcus ureæ*, which frequently occurs in urine, induces ammoniacal fermentation. Other species are found in fermenting wine and in a great variety of putrefying substances.

The so-called *pathogenic micrococci* belong, as a rule, among the minuter forms, and morphological distinctions between the species are as yet, for the most part, wanting.

Micrococci have been found at the seat of lesion or in various parts of the body in *diphtheria, scarlet fever, erysipelas, ulcerative endocarditis, cerebro-spinal meningitis, pneumonia, osteo-myelitis,* and *periostitis,* *pyæmia, puerperal fever, gonorrhœa,* and in connection with various phases of *suppurative inflammation.*

Their occurrence and significance will be considered in the descriptions of the lesions of these diseases in the section of this book devoted to Special Pathology.

Micrococci have been described as occurring in connection with a large number of other diseases, but without proof of their causative relation to them.

Another genus of sphero-bacteria is *Sarcina,* which embraces several species. Among these is the *S. ventriculi,* consisting of large cells usually grouped in clusters of four to sixteen. They are found in the stomach, both in health and disease, but are not proven to stand in a causative relation to the diseased conditions in which they occur.

II. ROD-SHAPED OR FILIFORM BACTERIA.

The most common and important genus in this group is called *Bacillus.* Some of the bacilli are short, and are apt to be grouped in pairs (see Fig. 23).

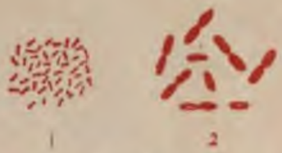

FIG. 23.—BACILLI IN PAIRS.
1 was formerly called Bacterium termo, and 2 B. lineola.

Some are long and may form chains or threads (see Fig. 24); some are slightly curved, and when joined together end to end, as is apt to occur in their growth, they form spirals (Fig 25).

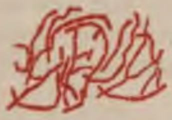

FIG. 24.

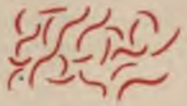

FIG. 25.

FIG. 24.—BACILLI ARRANGED END TO END, FORMING CHAINS.
Cultivated from laboratory air.

FIG. 25.—CURVED BACILLI FORMING SPIRAL CHAINS.
Spirillum tyrogenum.

Some of the bacilli are immobile; others are capable of performing the most varied movements when suspended in fluids. Many of the bacilli are concerned in the ordinary processes of decay which we call putrefaction, and are entirely harmless when introduced into the human or animal body. Many produce brilliant colors, red, yellow, green, etc., when growing in large masses.

Certain bacilli are capable, when they gain access to the body, of inducing serious disease, and even death—*pathogenic bacilli.* Thus malignant pustule, tuberculosis, typhoid fever, leprosy, glanders, Asiatic cholera, have been proven to be caused by different species of bacilli, which will be considered later in this book under their appropriate headings.

Bacilli of septicæmia of mice.—Dr. Koch has described small bacilli —0.8 to 1 μ long—occurring sometimes in pairs, sometimes in chains. which are found as spores or as developed bacteria in a great variety of putrefying fluids. They may be readily cultivated on gelatin, and the inoculation of the purified organisms or of the putrefying fluids into

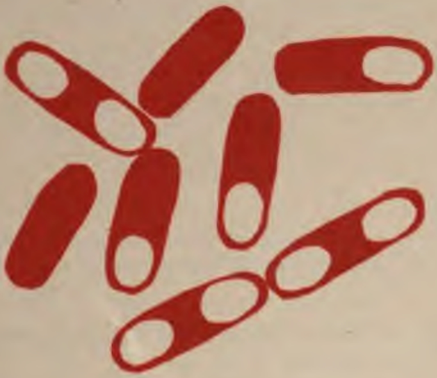

FIG. 26.—SCHEMATIC DRAWING OF BACILLI (B. MEGATERIUM) CONTAINING SPORES.

house mice produces symptoms characteristic of septicæmia. The bacilli may be found in great numbers in the leucocytes, in the subcutaneous tissue about the seat of inoculation, and very generally in the blood-vessels. The animals may die, or, if they survive, exhibit a marked immunity from the characteristic effects of subsequent inoculations. Field mice are not affected by inoculation with these bacilli, and rabbits usually but slightly.

Bacilli of malignant œdema of mice, guinea-pigs, and rabbits.—These are from 3 to 3.5 μ long, rounded at the ends. They frequently occur in pairs, and may exhibit spontaneous movements. They occur in cultivated soil, in hay, putrefying fluids, and elsewhere. When introduced into the subcutaneous tissue of the above-named animals, they proliferate and produce a wide-spread œdema about the seat of inoculation. In the clear reddish, œdematous fluid multitudes of the bacilli may be found.

After death they may be found in the blood, in the juices of internal organs, or upon serous surfaces. It is very difficult to cultivate them outside of the body. A large number of other bacilli are known which cause disease when inoculated into animals.

Leptothrix.—There exist under almost all conditions in the mouth and about the junction of the teeth and gums, single or in larger or smaller masses, long, slender, filiform bacilli, usually without transverse divisions, which have received the general name *Leptothrix buccalis.* There are some reasons for supposing that they may be concerned in caries of the teeth, in connection with which they are often found. But beyond this fairly well-founded conjecture, we have no reason to suspect them to be of any pathogenic significance. They are frequently entangled among scattered or larger and smaller masses of micrococci (Fig. 27). Leptothrix is a general name only, and we do not know whether it represents a single genus or species, or whether it is made up of undeveloped forms of some varieties of bacillus.

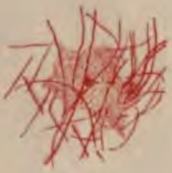

FIG. 27.—LEPTOTHRIX BUCCALIS WITH MICROCOCCUS COLONIES.
From the mouth of a healthy person.

III. SPIRAL-SHAPED BACTERIA.

The spirals of these cells may be close or open, few in number or numerous; some of them are ciliated at the ends. One of the common genera of these bacteria is *Spirillum.* *S. rugula* occurs in swamp water, on the surfaces of the teeth, and in fæces. It is thick, has open spirals, and occurs in chains or interlacing masses. Other species, among which may be mentioned *S. serpens* (Fig. 28), occur in stagnant water and in putrefying fluids.

Another important genus of spiro-bacteria is *Spirochæte,* whose cells are in general larger and more flexible than those of spirillum, with more closely set spirals.

Spirochæte denticola is found in the mucous membrane of the mouth and on the teeth mixed with leptothrix, and also in carious teeth. It is usually about 10 to 20 μ long, and pointed at the ends (Fig. 29). *S. plicatitis* is about 110 to 225 μ long, very slender, with many spirals, and blunt ends. It moves with great rapidity, and is frequently found in summer in swamp water and in eave-troughs.

One of the more important species of this group is that causing Relapsing Fever, under which heading it will be described.

It should always be borne in mind, in examining tissues taken from the dead body, that various forms of bacteria commence to proliferate in the fluids and tissues very soon after death, and may develop in extraordinary numbers in a very short time.

The bacteria are often divided into two great groups depending upon the conditions under which they live. Those which nourish themselves on dead organic material are called *saprophytes* or *saprophytic bacteria;* those which feed upon living organisms, *parasites* or *parasitic bacteria.*

While some of the parasitic bacteria are always parasitic (*obligatory parasites*), there are others which are only occasionally parasitic; these are called *facultative parasites.* There are others, furthermore, which usually are parasitic, but occasionally live as saprophytes; these are *facultative saprophytes.*

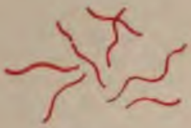

FIG. 28.

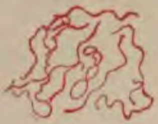

FIG. 29.

FIG. 28.—SPIRILLUM SERPENS.
From putrefying hay infusion.

FIG. 29.—SPIROCHÆTE DENTICOLA.
From mouth of healthy person.

This classification is by no means an exact one, but it serves a useful purpose in indicating in a general way the favorite life conditions of different forms of bacteria.

The Relations of Bacteria to Disease.—Bacteria of various forms may occur on the surface of the skin and mucous membranes, and in the lungs, exerting no apparent influence whatever, so far as we can judge by morphological appearances, upon the surrounding parts. They may occur on the surface of wounds, ulcers, etc., without the production of any evident changes; they sometimes lie within cells which appear otherwise perfectly normal. On the other hand, the cells and tissues in their vicinity may show very marked alterations, which are presumably due to their influence. The cells may be swollen, their nuclei may break down or disappear, and the protoplasm may be converted into a mass of shining or coarsely granular particles, or may completely disintegrate. The intercellular substance near the bacteria may also soften and disintegrate. In a word, the tissue in their immediate vicinity is often found in a condition of necrosis of one kind or another. The walls of blood-vessels near which they lie may die, and the blood which they carry may form thrombi. The bacteria may themselves enter the vessels and proliferate in the blood; they may be swept away as emboli to remote parts of the body (Fig. 30), and establish new foci of bacterial proliferation and tissue necrosis. Some forms, instead of causing the immediate death of the tissue, appear to incite inflammatory changes about themselves (Fig. 31). These may be simple, and similar to those produced by the presence of any irritating foreign body; or it would seem that the bacteria may determine, in some way as yet unknown to us, very peculiar and characteristic inflammatory changes, which may result in the formation of new tissues of various kinds. Some forms of bacteria find in the blood, others in the lymph spaces and vessels, the conditions most favorable for their proliferation.

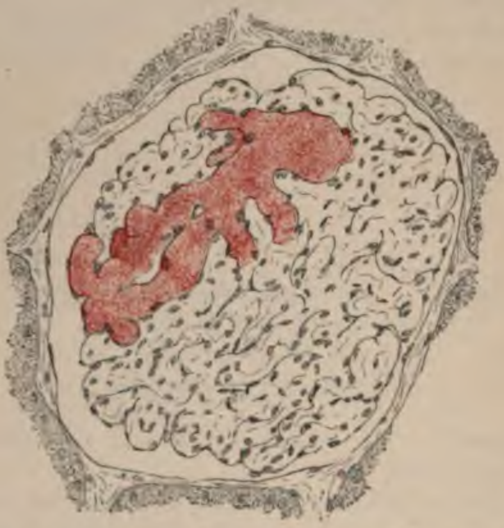

FIG. 30.—BACTERIAL EMBOLUS IN THE BLOOD-VESSELS OF THE GLOMERULUS OF THE KIDNEY IN MALIGNANT ULCERATIVE ENDOCARDITIS.

As the bacteria grow in the tissues or fluids of the body, they form and set free various kinds of chemical compounds. These are called ptomaïnes; and some of these ptomaïnes are very poisonous indeed. It seems at present highly probable that a large proportion of the deleterious effects which pathogenic bacteria may cause as they grow in the body are due to the ptomaïnes which they elaborate and set free.

It will be seen, from what has now been said of the bacteria, that in different parts of the system in health, and in a large number of diseased conditions, various forms of bacteria occur; but it is quite evident that the significance which we must attach to their mere presence varies greatly. In a large number of cases, especially when on parts exposed to the air or in the gastro-intestinal canal, they are evidently of no more importance than so much inorganic dust. When, however, special forms of bacteria are found to occur uniformly in connection with well-defined diseases, or in their lesions, the conjecture is certainly justified that they may have something to do with their production. Yet in all such cases we have to consider the possibility that it is the diseased state or the character of the lesion, produced perhaps by other causes, which affords conditions suitable for bacterial growth, and that they may consequently occur in considerable numbers; while in the absence of these conditions they would be unable to develop. Even the constant occurrence in the body, in certain diseases, of bacteria which evidently produce well-marked local effects, either inflammatory or degenerative, does not absolutely prove their causative relation to the disease, although it renders it to a certain degree probable.

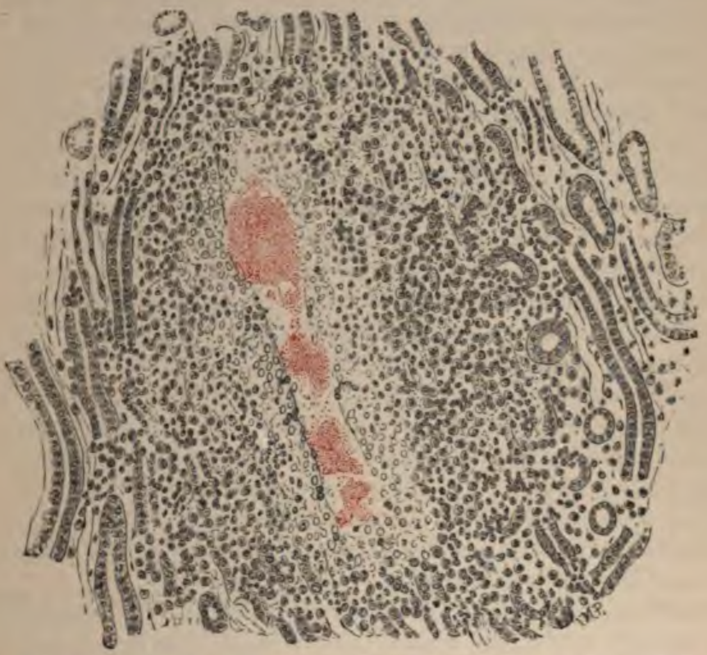

FIG. 31.—COLONIES OF MICROCOCCI IN A BLOOD-VESSEL OF THE KIDNEY CAUSING A SMALL ABSCESS. From a case of pyæmia.

Around the dilated and partially necrotic blood-vessel in which the bacteria lie is an area of necrotic tissue and small celled infiltration or zone of pus.

In order to absolutely prove the bacterial origin of an infectious disease, we must not only be able to demonstrate the constant presence in the body at some time of a special form of bacteria, but we must obtain them in an absolutely pure condition, unmixed with any other living thing, or with any chemical substance not belonging to them, and, by the introduction of the purified organisms into a healthy animal, be able to produce the disease in some definite form. When all this is done, and not before, can we assert with absolute certainty the causative relation between a given form of bactèria and any special infectious disease. But the fulfilment of these strict requirements is very difficult in many cases, and in some, apparently, almost if not quite impossible; for we must remember, in the first place, that the lower animals, upon which alone, for the most part, inoculation experiments are practicable, are apparently not subject to certain important diseases of men; and second, that they present among themselves the most marked differences in the degree and manner in which they are affected by the inoculation of pathogenic bacteria. Desirable as is the complete fulfilment of the above requirements in every case, it must be admitted that a reasonable certainty regarding the bacterial origin of a given disease may be arrived at without positive results from the inoculation of the bacteria associated with its lesions.

The discussion of the probabilities of the bacterial origin of certain classes of disease, and the long series of phenomena exhibited by them, which the bacterial theory very satisfactorily explains, does not fall within the scope of this book.

The complete demonstration which certainty requires has as yet been furnished in but a very small number of cases. In a considerable number of other cases, although the complete cycle of proof has not yet been established, enough has been done in the way of study and experimentation to render it altogether probable that certain forms of bacteria are the cause of the diseases.

Conditions Influencing the Occurrence of Bacterial Diseases.—It has been learned, as the result of a great deal of observation and experiment, that, although certain diseases are invariably caused by the presence and growth in the body of particular species of bacteria, and never occur without them, still there are various other accessory factors which have a very important bearing upon the inception and course of the diseases. Thus, while the presence in the body of a particular species of bacteria is the most significant and fundamental of the determining agencies in the bacterial diseases, the numbers in which they are present—*i.e.*, the size of dose—and the varying virulence which the same species under different conditions possesses, as well as the varying capacity of resistance to the incursions of the germs which the body cells at different times and under differing conditions exhibit, are all factors of the greatest moment.

It should be always borne in mind that the human body is a great aggregate of groups of co-ordinated cells which, under normal conditions, all act in harmony for the maintenance of the life and functions of the individual. The cells and cell communities in health not only do this, but they have the power of resisting and to a certain extent overcoming the various deleterious agencies to which the body is more or less constantly liable.

What we call hereditary or acquired predisposition to a bacterial disease, such as tuberculosis, for example, is simply a lack of the usual capacity of the cells of the body—whether through a structural or physiological fault we do not yet know—to cope with the destructive tendencies of the living bacteria when once they gain a foothold in the body.

We thus see that, in studying the conditions under which bacterial diseases occur, the work is by no means complete when we have demonstrated the bacterial species which causes the disease, but that then the more obscure determining and influencing agencies must be worked out in each particular case.

Phagocytes.—It is well known that the introduction into the body even of extremely virulent species of bacteria does not always cause disease. It is furthermore established that when recovery from a bacterial disease occurs, the germs after a time disappear from the body. These well-known facts show either that the body possesses the power of directly destroying certain bacteria, or of furnishing conditions which are so unfavorable for their life that they die.

It is believed by many that certain cells of the body are capable of taking up bacteria, which get into the tissues, into their protoplasm, and there destroying and perhaps digesting them, and that thus the destruction of bacteria in the body may be brought about.

Others believe that certain of the body cells, the leucocytes, for example, which are often found gathered about masses of bacteria in the body, cut off the supply of oxygen or nutriment from the germs and thus cause their destruction.

The cells which take up into their bodies the bacteria, as well as other foreign bodies, are called Phagocytes. This assumed mode of destruction of bacteria forms a most suggestive and fascinating hypothesis, but its significance and importance are not yet very fully demonstrated.

The reasons for believing that certain well-known and important diseases are caused by bacteria, as well as a description of the particular organisms concerned, will be found in that part of this book which treats in detail of the lesions of various parts of the body or of the lesions of the general diseases.

METHODS OF STUDYING BACTERIA.

The simplest mode of studying bacteria is to examine them either in the fluids in which they lie, or in thin sections of the tissues mounted in one-half-per-cent salt solution, and for the study of many of the phenomena of life this method is important. The tissue and other elements, however, with which they are often associated, greatly interfere with the study of the bacteria, and many forms are so small as to be scarcely recognizable in the natural condition.

By far the most important aid in the morphological study of the bacteria is derived from the use of staining agents. Most of the bacteria are stained more or less readily by one or more of the aniline dyes. The ease with which they are colored varies considerably in different species and with the different dyes. The tissue elements, and a variety of other materials with which the bacteria may be associated, also stain more or less readily at the same time; but most of these part with their color more readily than do the bacteria, on being treated with alcohol or dilute acids. We are thus enabled to obtain a differentiation in color between bacteria and other structures. The bacteria, moreover, differ among themselves in respect to the tenacity with which they hold their stain in the presence of decolorizing agents; and upon this fact is based one of the important methods of distinguishing between different species.

Among the aniline dyes more commonly employed for bacteria staining may be mentioned fuchsin, gentian violet, methylene blue, and Bismark brown. Of these, the fuchsin and gentian violet are the best for ordinary purposes.

A saturated alcoholic solution of these dyes should be kept in a tightly stoppered bottle, and from this the more dilute solutions required for staining may be prepared. For ordinary purposes, one part of the alcoholic solution of fuchsin or gentian violet, added to twenty parts of water, will give a staining solution of suitable strength. This should be prepared in small quantities as required, since it does not keep well, and a granular precipitate is apt to form in a few days.

Special stains and modes of staining, such as are necessary for some forms of bacteria, such as the tubercle and typhoid bacillus, will be described under the appropriate headings. We are speaking here only of the general methods.

To Stain Bacteria in Fluids.—A small drop of the fluid is placed on a cover-glass, spread a little with a needle, and allowed to dry by evaporation in the air or by gentle heating high over a flame. The cover-glass is now held with the forceps, and—specimen side up—passed moderately rapidly three times through the flame of an alcohol lamp or a Bunsen burner. The material on the cover should not be burned. This heating not only fixes the contents of the fluid firmly on to the glass, so that it will not easily soak off, but it renders insoluble any albuminous materials which may be mixed with the bacteria. and which might otherwise interfere with subsequent examinations, by forming granular precipitates.

A drop of the aqueous staining fluid is now put on to the dried specimen on the cover-glass, and if this be held in the forceps and tilted slightly up and down a few times, so as to bring fresh portions of the staining fluid into contact with the bacteria, the staining will usually be completed in two or three minutes. The stain is now washed off with a jet of water from the wash-bottle, and the specimen is either mounted directly in a drop of water for temporary study, or the washing-water is drained off, and after drying in the air is mounted directly in balsam.

It is well to use balsam which has been softened, when this is necessary, with oil of cedar or xylol, since chloroform is apt to decolorize the bacteria.

If the bacteria which are to be stained are in solid masses, as is apt to be the case in pure cultures on solid media (see page 99), a small drop of distilled water should be first put on the middle of the cover-glass, and a very minute quantity of the bacteria mass rubbed into it with a needle, and then dried and stained as before.

To Stain Bacteria in Tissues.—The tissues should be well hardened in alcohol. Thin sections are placed in the above-described coloring solutions, where they may remain from one to ten hours. Gentle warming (40° to 50° C.) will hasten the staining. The entire tissue as well as the bacteria is in this way deeply colored The sections are rinsed with distilled water and then placed in alcohol. This, with varying degrees of rapidity with different stains and tissues, gradually extracts the color from the tissue, most slowly from the nuclei. The time required and the exact degree of decolorization to be sought for must be learned by experience in different cases. Sometimes five, sometimes thirty minutes are required. It is often necessary, and the decolorizing of the tissue is thereby hastened, to add a few drops of acetic acid to the alcohol. When acetic acid is used, it should be finally thoroughly washed out by alcohol. The specimens are now cleared up by oil of cloves and mounted in balsam. Oil of cloves removes the color from some forms of bacteria, and in this case xylol or oil of bergamot should be substituted for it. In specimens prepared in the above way, the nuclei of cells usually retain to some extent a color similar to that of the bacteria, but their size and shape serve for the differentiation.

A much more generally useful method of staining bacteria in the tissues than that just given, although not in all cases applicable, is that known as *Gram's method.* The tissues from which the sections are made should have been hardened and preserved in alcohol. The sections are stained for from two to four minutes in *aniline-gentian-violet solution.* This is prepared as follows: 5 c.c. aniline oil are added to 100 c.c. distilled water; this mixture is well shaken, and the excess of aniline oil is filtered off through a moistened paper filter. To the clear filtrate saturated alcoholic solution of gentian violet is added until the fluid becomes opalescent (about 1 part of the dye to 10 of the water will usually be enough). A small quantity only of this fluid should be prepared at once, as it does not keep very well.

From the staining solution the sections are transferred directly to a solution of iodine in potassium iodide and water (I 1.0—KI 2.0—H_2O 300.0). In this they remain from one to three minutes, a precipitate forming in the solution and the sections becoming of a dark reddish or slate color. The sections are now transferred to absolute alcohol, which should be changed two or three times so as to dehydrate the specimen, which at the same time will lose much of its color. Finally the decolorization is completed and the section cleared up by oil of cloves, and it may then be mounted in balsam. Very little color should be visible to the naked eye in the specimen when it is ready to mount. If the specimen have been imbedded in celloidin for the purpose of section-cutting, this will be dissolved from the sections by the oil of cloves. Then it is well to use a second portion of oil of cloves, so as to get rid of the superfluous half-dissolved celloidin. It is also well to tint the oil of cloves lightly with a few drops of alcoholic solution of eosin, and then the violet-colored bacteria will stand out in sharp contrast with the reddish tissue elements.

It should always be borne in mind in staining the bacteria that great exactness is not necessary either in preparing the coloring solutions or in the time of exposure of the bacteria to them. We are seeking for certain effects—namely, the staining of the germs—and this depends not only upon the quality and strength of the dye, the time of exposure, etc., but also upon the nature of the bacterial species and its condition at the time the staining is attempted. Thus it not infrequently happens that bacteria which will stain readily and deeply with a given solution when they are in a condition of active growth, may scarcely be at all colored if they have been dead or inactive for a long time, although their outward shape appears to be unchanged. So it should be remembered that, while there is little difficulty in most cases in staining the bacteria, the operation is not one of mere routine, but requires intelligent attention to the particular conditions of the species in hand.

The Microscope.—For the recognition and study of bacteria, especially of the minuter forms, the best optical apparatus is requisite.

Good dry lenses will answer in many cases; but for finer work homogeneous immersion lenses (at least one-twelfth) must be employed.

A special mode of illumination is also in many cases requisite, such as may be obtained by the use of the Abbe condenser. The structural outlines which the tissue elements present, in many cases greatly interfere with the observation of the color by which the stained bacteria are recognized. This interference is considerably reduced by mounting the specimens in balsam; but usually not sufficiently so for the study of the minuter forms. The Abbe condenser is a combination of lenses, placed close beneath the object, by means of which, owing to the direction in which the rays of light are brought upon the object, structural outlines are made to nearly entirely disappear, and in this way the stained objects, the bacteria, stand out much more distinctly than would otherwise be the case against the nearly homogeneous background.

Artificial Cultivation of Bacteria.—For the complete investigation of the different forms of bacteria, particularly in their relations to disease, we must study their life history and the effects of their inoculation into healthy animals. It has long been known that bacteria could be cultivated in a variety of artificially compounded, so-called nutrient media or soils. Fluids were formerly used for this purpose, but it is very difficult to separate single species in fluid media, and to detect contaminations when they occur. Moreover, the inevitable mechanical disturbances of the fluid prevent, for the most part, the formation of gross characteristic appearances in the masses of growing bacteria. Dr. Robert Koch introduced, a few years ago, a technical improvement of inestimable value, in suggesting and formulating the details of using solid media for the cultivation of bacteria. Among these may be mentioned sterilized boiled potatoes and gelatinized infusions of various natural or artificially compounded substances sterilized by heat. Different species of bacteria often require different nutrient media, and some require different temperatures for their most flourishing growth. They usually grow on the surface of the nutrient media in sharply circumscribed masses, and different species may grow side by side in the same receptacle for considerable periods, without in the slightest degree interfering with one another or tending to mix. The mode of growth and general appearances of the proliferating bacterial masses on the solid medium often present very characteristic differences between different forms, and thus not only furnish valuable means of identifying species, but render possible an early detection of contamination from chance admixture of species. A given species of bacteria may be cultivated through a series of generations by transferring, with proper precautions, a minute portion from a growing colony to a fresh surface of sterilized soil. After cultivation through several generations, the species may be presumed, and by microscopical examinations proved, to be entirely pure, and the effects, if any, produced by its inoculation into healthy animals, to be due to it alone.

The Preparation and Use of Culture Substances.—There are many culture media, some of which are best suited for one, some for another species of bacteria. Those most commonly used are beef-tea, beef-tea rendered solid by gelatin or agar-agar (called "nutrient gelatin," or "nutrient agar"), boiled potatoes, coagulated blood serum, and milk.

Potatoes.—The potato is scrubbed with a brush under the water faucet and the ends cut off with a knife, leaving a segment about an inch and a half long. With a tin cylinder about an inch in diameter, made like an apple corer, a cylinder is cut lengthwise out of the potato segment, and this is divided lengthwise with the knife into two oblique sections. These pieces of potato are now placed, the narrow end up, in large test tubes about five inches long and a little more than an inch wide, which have been plugged at the mouth with cotton, and freed from all germs which might have been upon or in them, *i.e.*, sterilized by heating in an oven for an hour at 160° C. Several of the potato tubes thus prepared are now put together into an open-wire basket and steamed for an hour, and again for an hour on the following day. They are now ready to be sown with the bacteria on the oblique surfaces (see Fig. 32).

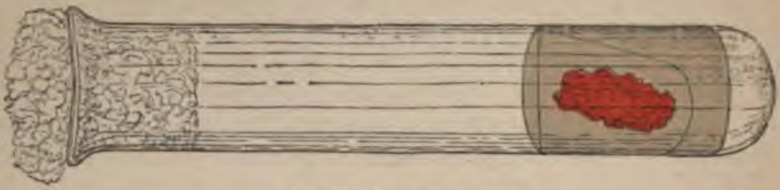
FIG. 32.—A POTATO CULTURE.

Growing in a sterilized tube plugged with cotton. The red mass or "colony" on the oblique surface of the potato is made up of myriads of bacteria.

Nutrient Gelatin.—One pound of lean beef is chopped fine, stirred into one litre of water, covered, and set away in the refrigerator for twelve hours. The red fluid is now completely separated from the meat by squeezing through a cloth into an enamelled saucepan, which fits into a larger vessel serving as a water-bath. To the beef juice are added ten per cent of clear French gelatin, one per cent beef-peptone, and one-half per cent of common salt. The mixture is now heated in the water-bath until the gelatin is dissolved, when it is carefully neutralized by the addition of a sufficient quantity of an aqueous solution of sodium carbonate. It should be made exactly neutral or *very slightly* alkaline. The white of two eggs well beaten is now added, and the whole boiled vigorously for half an hour. It is then filtered through a thick layer of sterilized cotton into a flask, and should form a perfectly clear, slightly yellowish mass which is quite firm and solid on cooling. It is now filled into sterilized test tubes which have been plugged with cotton before sterilizing—about two inches in depth of the material being put into each tube—and these are steamed for from twenty minutes to half an hour, and again for an equal period on the following day, when they are ready for use (see Fig. 33).

Nutrient Agar.—This is made and filled into tubes in the same way as the nutrient gelatin, save that one per cent of the agar is added in place of the gelatin. As the agar is less readily soluble than the gelatin, it will have to be boiled longer on the water-bath before neutralizing. After the last sterilization, the agar tubes are placed in a slanting position to cool, so that the agar may present a long, oblique surface.

The *Beef Tea* is made in the same way, save that no solidifying material is added, and it may be filtered through paper.

The ***Milk*** is prepared by filling it into test tubes (about the same amount as in the case of gelatin), and steaming for an hour on two successive days.

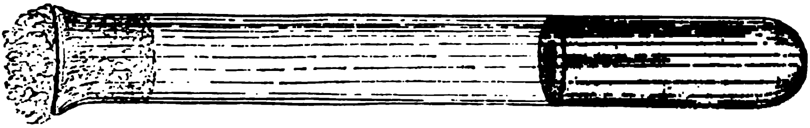

FIG 33.—A GELATIN TUBE CULTURE.

The bacterial growth is confined to the seat of inoculation, *i.e.*, the surface and the line of needle puncture below.

The mode of preparing blood serum need not be considered here.

Having thus seen how the nutrient media are prepared, let us see briefly how they are used in studying the bacteria.

In the first place, it is necessary to get from the various mixtures of several species, as they apt to occur in nature or in diseased parts, single species growing by themselves, so that their life history and characters may be studied in detail. To show by an example how this is done, we will suppose that we have a sample of water containing bacteria, and wish to learn how many there are and of what species, and to get them into separate receptacles for study. We melt the gelatin in one of the test tubes which we know to contain no living bacteria—because we have sterilized both the tube and its contents by heat—and add to it a measured volume, usually one c.c., of the water, and mix them by shaking; we now take a glass plate which has been sterilized by heat, lay it upon a cold surface, and pour out the mixture of water and nutrient gelatin in a thin layer upon it. When the gelatin solidifies, the invisible germs which the water contained are caught and held in position by it, and if the whole be now set away in a sufficiently warm place the living bacteria will presently commence to grow.

After a few hours or days, from each one of the single living bacteria scattered through the gelatin so many new germs may have developed that they form a mass, called a colony, large enough to be visible to the naked eye. As different species grow in different ways, some forming colored colonies, some fluidifying the gelatin, some growing much more rapidly than others (see Fig. 34), we can usually recognize the different species either with the naked eye or under the microscope, and with a fine sterilized needle can pick out portions of the different colonies and transfer them to the tubes of nutrient media of one kind or another which we have prepared, and study their growth there in the form of pure cultures.

The transfer to the tubes is made by plunging the needle which has touched the plate colonies down into the gelatin or agar, or drawing it over the surface of the potato. This is called inoculating the culture media.

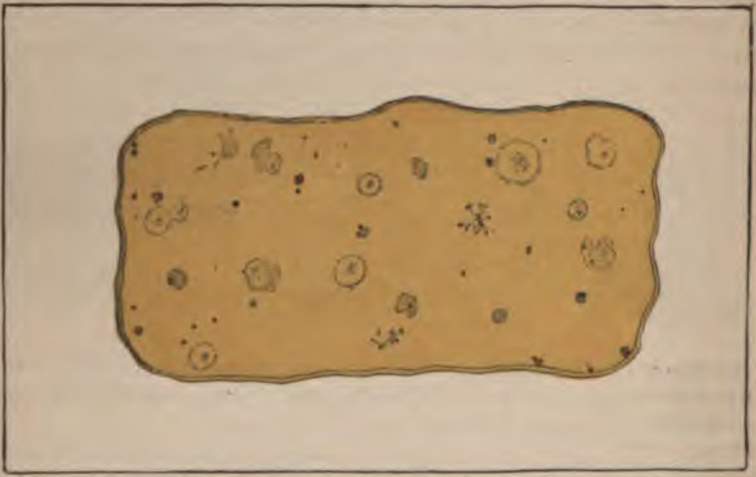

FIG. 34.—A GELATIN PLATE CULTURE.

Showing bacterial colonies of various species. This plate was made by mixing 1 c.c. of the ordinary drinking water of New York City with the nutrient gelatin, and allowing the bacteria to grow for two days.

By the use of this principle of the plate culture, sometimes with one form of culture medium, sometimes with another, and with various modifications of the technique, we can separate the bacteria which occur in the body in disease, and, by combining animal inoculations with our other observations, can collect the data which enable us to decide whether the bacteria which are associated with certain lesions actually cause them.

The most scrupulous care is required in sterilizing the nutrient media and the utensils and instruments used, and the greatest caution should be exercised, in transferring the bacteria from one receptacle to another, to prevent contamination. A large experience in this sort of manipulation is necessary before reliable results can be obtained in original investigation, since the slightest error or carelessness in manipulation, or failure to observe the occurrence of contamination, is liable to entirely vitiate the results of long series of experiments. It is only by an extended preliminary training in the cultivation of some of the more characteristic and easily recognizable forms, under a variety of conditions, in a perfectly pure state, through a series of generations, that one can be assured of his capacity to carry on researches in this most difficult and intricate field.

The methods of inoculation of animals with pure cultures, and the precautions to be observed, as well as a description of the various forms of apparatus made use of in practical bacteriology, must be sought in more extended treatises on this subject.

It is wiser for one purposing to carry on bacterial researches to gain a practical acquaintance with methods and apparatus in a well-appointed laboratory, than to make the attempt to work out the methods from books.

Material obtained from the human body which is to be subjected to bacterial examination should be collected with every precaution against accidental contamination: if fluid, in sterilized vessels; if solid, wrapped in cloths saturated with 1:1,000 corrosive sublimate solution, and brought at the earliest possible moment to the laboratory.[1]

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[1] *Bibliography.*—The most useful work for general purposes is that by *Flügge* on "Micro-organisms," 1886, in which will be found an excellent bibliography. The most complete treatise on bacterial technology is *Hueppe's* "Methoden der Bakterienforschung," 1889. Most valuable also are the publications of *Koch*, among which especially to be noted are "Wundinfectionskrankheiten," Leipzig, 1878; English translation, "Traumatic Infective Diseases," Sydenham Soc., 1880; and "Mittheilung . d. k. Gesundheitsamte," Berlin, 1881, vol. i. Among the general works in English may be mentioned the work of *Sternberg* and *Magnin* on "Bacteria," which contains a bibliography and general description of various forms of bacteria, with micro-photographs, modes of staining, etc. *Crookshank's* "Manual of Bacteriology" contains fine illustrations of apparatus and cultures, and many formulæ.

INFLAMMATION.

The condition of our knowledge is not such as to admit of an exact definition of the term inflammation; nor is there an entire agreement concerning the number of morbid changes to which this term may be properly applied. We have to be contented with describing the conditions which are probably of an inflammatory character.

I. EXUDATIVE INFLAMMATION.

We employ this term to designate a form of inflammation characterized by the presence of an exudate derived from the blood-vessels. The vessels contain an increased quantity of blood. The plasma, the white cells, and the red cells escape from them and are found in the tissues. A portion of tissue in which such an inflammation is going on is, therefore, naturally congested, swollen, hot, and tender.

The different stages of such an exudative inflammation can be observed in animals during life.

The transparent connective-tissue membranes of some of the lower animals can be studied under the microscope while inflammatory changes are going on. In this way we have learned that there is first a dilatation of the arteries, veins, and capillaries, and an increased rapidity of the circulation of the blood. Then the blood-current becomes slower; white blood-cells accumulate in the small veins and capillaries, and adhere to their walls. Then the white cells, changing their shape, find their way between the endothelial cells of the vessels, through their walls, and appear on the outside of the vessels in the tissue (Fig. 35). This process is called *emigration*. Red blood-cells in smaller numbers may also pass through the walls of the capillaries and veins, and this is called *diapedesis*. At the same time the plasma of the blood transudes through the walls of the vessels and infiltrates the tissues as serum; while, by the union of substances contained in the blood plasma and in the white cells, fibrin is formed.

In this simple manner are elaborated the inflammatory products—pus, serum, and fibrin. The pus cells are emigrated white blood-cells; the serum is part of the plasma of the blood; the fibrin is produced by a union of the fibrinogen in solution in the blood plasma with substances contained in the white blood-cells, and appears coagulated in the form of granules, amorphous masses, or a reticulum.

In order that these inflammatory products may be formed, it is evidently necessary that the blood should continue to circulate through the vessels of the inflamed tissue.

Different examples of this form of inflammation give different amounts of inflammatory products, and also different relative proportions of serum, fibrin, and pus; while extravasated red blood-cells may also be intermingled with them.

The inflammatory products may accumulate wherever there are cavities to receive them, in the interstices of connective tissue, on the surfaces of the serous membranes, and in the serous cavities.

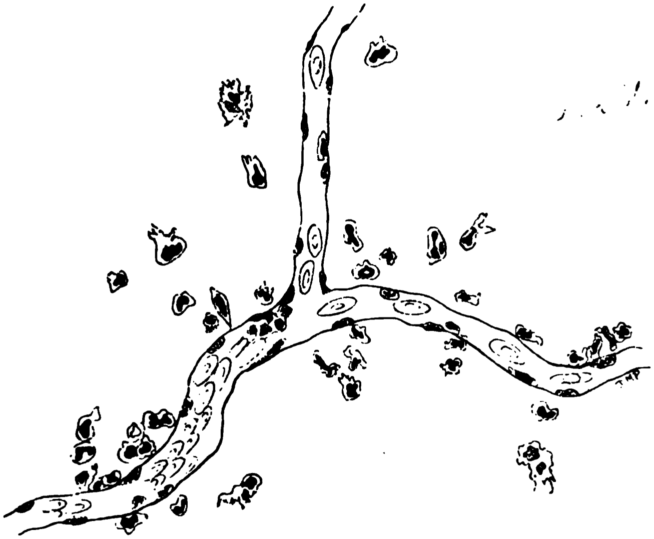

FIG. 35.—EMIGRATION OF WHITE BLOOD-CELLS IN INFLAMED BLADDER OF FROG.

The simplest form of exudative inflammation is that in which the changes in the blood-vessels and the production of serum, fibrin, and pus are associated with few or no changes in the surrounding tissues.

Such an inflammation in *connective tissue* produces congestion, swelling, and infiltration, with serum, fibrin, and pus.

In the ***mucous membranes*** there are congestion, swelling, emigration of white blood-cells, diapedesis of red blood-cells, and transudation of serum; the quantity of the inflammatory products is usually small. The production of mucus is at first arrested, afterwards increased. There may be increased desquamation of epithelium and the formation of superficial ulcers. When it occurs in the mucous membranes, such a simple exudative inflammation is usually called "acute catarrhal inflammation" (Fig. 36).

In the *viscera* there are more or less congestion and swelling; the quantity of inflammatory products is moderate; the functions of the viscus are apt to be deranged.

The inflammation is of the same nature in connective tissue, the mucous membranes, and the viscera. The differences are due to the different structures of these parts. The process is always a transitory one, and does not change the tissues of the inflamed part. After the termination of the inflammation, the congestion subsides, the inflammatory products are absorbed, and the parts return to their natural condition.

A somewhat different character is given to the exudative inflammation if there is an excessive emigration of white blood-cells. This is often called "purulent or suppurative inflammation." The excessive quantity of pus cells may or may not be accompanied by serum and fibrin. This is of more severe type than is a simple exudative inflammation. Connective tissue is simply infiltrated with the inflammatory products. In the mucous membranes, the pus cells are mixed with the mucus on the surface of the inflamed membrane. In the viscera, the stroma is infiltrated with the pus cells (Fig. 31).

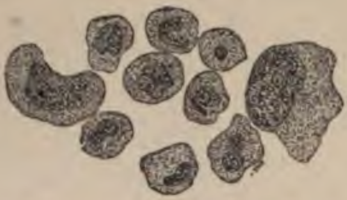

FIG. 36.—PUS CELLS FROM CATARRHAL INFLAMMATION OF BRONCHIAL MUCOUS MEMBRANE.

Exudative inflammation may be accompanied by the degeneration and death of tissue. The changes in the blood-vessels and the production of serum, fibrin, and pus are developed in their ordinary way, but there is added degeneration or death of portions of the tissues in which the inflammation is situated.

Suppurative inflammation is due, in the majority of cases, to the presence and growth in the tissues of bacteria. The most common forms of bacteria found in connection with it are the *Staphylococcus pyogenes* aureus and albus, and the *Streptococcus pyogenes*. They frequently occur together in the same inflammatory process. In general, it may be said that while the staphylococcus tends to cause localized suppurative inflammations and abscesses, the streptococcus is more apt to induce progressive forms of inflammation and to give rise to general infection of the body.

The staphylococcus pyogenes aureus is widely distributed in the air and among dust particles, particularly in dirty inhabited regions. It is not readily killed by drying or freezing. It grows readily in various artificial culture media, assuming, when exposed to the air, a golden yellow color. It fluidifies gelatin. Staphylococcus pyogenes albus appears to be simply a variety of staphylococcus, differing from the staphylococcus pyogenes aureus especially in the absence of color in the growths.

The streptococcus pyogenes grows more slowly than the staphylococcus, does not fluidify gelatin, develops no color. The chain form is apt to be most marked in beef-tea cultures. There is much reason for believing that the streptococcus pyogenes is identical with the streptococcus of erysipelas and diphtheria.

In *connective tissue* there are congestion, exudation of serum, emigration of white blood-cells, and death of portions of tissue. The dead tissue softens, breaks down, and cavities are formed which contain serum, pus cells, and fragments of dead tissue. Such cavities, with their contents, are called "abscesses." When healing occurs, the cavities of the abscesses are filled, first with granulation tissue, and afterwards with cicatricial tissue.

In *mucous membranes* there are congestion, exudation of serum rich in fibrino-plastic substances, emigration of white blood-cells, and necrosis of tissue. The fibrin infiltrates the stroma and coagulates on the surface of the mucous membrane, so as to form false membranes. The pus cells are entangled in the fibrin. The necrosis involves only the epithelium, which passes into the condition of coagulation necrosis and forms part of the false membranes; or it involves the stroma also. The death of the epithelium forms superficial erosions; that of the stroma, ulcers of varying size and depth. Such an exudative inflammation of the mucous membranes is called "croupous" or "diphtheritic" (Fig. 37).

In the mucous membranes, when healing occurs, the dead epithelial cells may be replaced by new cells of the same kind, but the ulcers formed by the death of the stroma have to be filled by granulation tissue.

In the *viscera*, the congestion is more or less marked, and there are exudation of albuminous serum and emigration of white blood-cells. At the same time the cells of the viscus undergo degenerative changes; they are swollen, or coarsely granular, or broken down. There may also be death of portions of the stroma, with groups of cells and the formation of abscesses. In recovery, the degenerated cells may be replaced by new visceral cells, and the abscess cavities are filled by granulation tissue.

This variety of exudative inflammation is of severe type, is accompanied with marked symptoms, and, after it has subsided, leaves changes in the tissues.

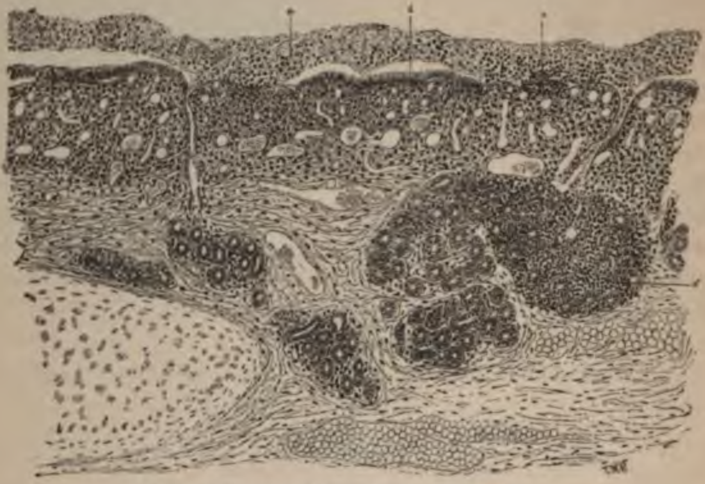

FIG. 37.—CROUPOUS INFLAMMATION OF TRACHEA.

n this case there is purulent infiltration of the mucosa and submucosa, and of portions of the mucous glands. *a*, false membrane ; *b*, portion of intact epithelium ; *c*, infiltration of the mucosa with fibrin ; *d*, portion of mucous gland infiltrated with pus.

Exudative inflammation may be accompanied or followed by the production of new tissue. The congestion and the exudation of serum, fibrin, and pus are well marked, but from the very first there is also a

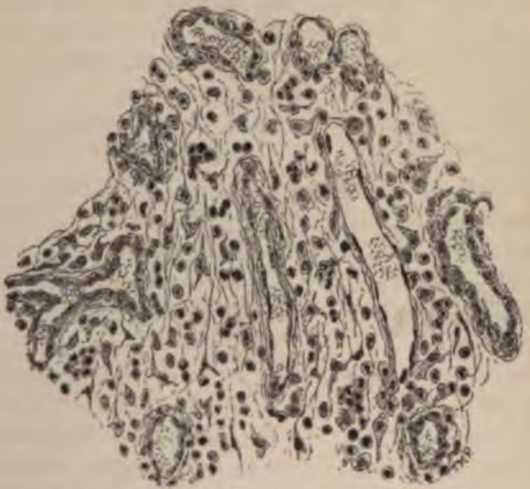

FIG. 38.—GRANULATION TISSUE FROM WOUND OF SKIN.

formation of new tissue. This new tissue is composed simply of small cells with a scanty basement substance between them—indifferent tissue. Or it is composed of cells like the white cells of the blood, small polygonal cells with a large nucleus, fusiform and branched cells, large epithelioid cells, a basement substance and blood-vessels—granulation tissue (Fig. 38).

The basement substance is at first scanty and homogeneous or finely granular. As the granulation tissue grows older, it becomes more abundant, denser, and fibrillated.

The blood-vessels are very numerous and at first thin-walled. They are formed from the original blood-vessels of the connective tissue by the outgrowth of solid sprouts of protoplasm which afterwards become channelled, so that the blood passes into them, and changed so as to form a wall of endothelial cells (Fig. 39).

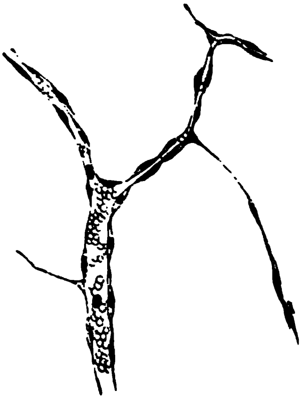

FIG. 39.

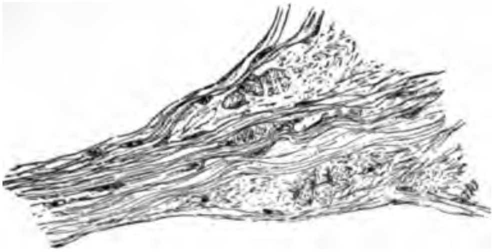

FIG. 40.

FIG. 39.—DEVELOPING BLOOD-VESSELS IN NEW-FORMED TISSUES.
FIG. 40.—CICATRICIAL TISSUE.

The surface of such granulation tissue is dry, or coated with serum, pus, or fibrin.

It may remain in the condition of granulation tissue for an indefinite length of time, or it may be gradually changed into cicatricial connective tissue. In the latter case, the basement substance increases and becomes dense, the cells disappear, the new blood-vessels are obliterated, and there results a dense, contracted area of connective tissue (Fig. 40).

In *connective tissue*, the fibrin, serum, and pus are found in varying quantities. The new tissue forms thickenings and adhesions.

In the *mucous membranes*, the serum, fibrin, and pus are scanty; the new tissue thickens the stroma of the membrane; the functions of the mucous glands may or may not be changed.

In the *viscera*, the quantity of the inflammatory product varies. The new tissue is produced in the stroma; there may also be degeneration of the visceral cells. In the viscera, this may be called a "diffuse inflammation."

The most marked features of this form of exudative inflammation are its tendency to persist for a long time, the frequency with which it runs a subacute course, and the permanent character of the new tissue produced.

II. PRODUCTIVE INFLAMMATION.

This term is used to designate a form of inflammation of which the product is new connective tissue, while congestion and exudation are nearly or entirely absent. Such an inflammation may be acute; it is more often chronic. Concerning the chronic forms opinions are not agreed. Many examples of this lesion are often called sclerosis, fibroid degeneration, or senile changes. In the present state of our knowledge, it seems to me better to include all these changes with the inflammations.

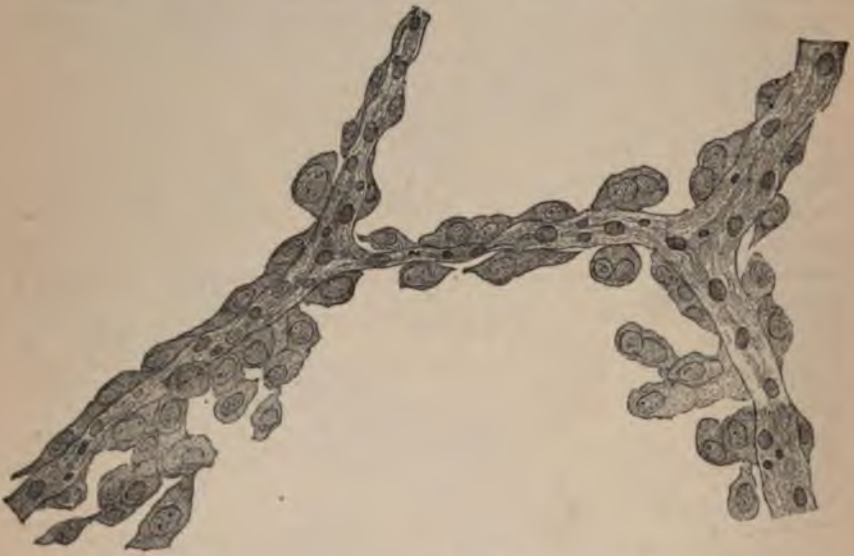

FIG. 41.—OMENTUM OF DOG, SHOWING CELLULAR PERITONITIS ON FOURTH DAY, × 500 and reduced.

In acute productive inflammation, or cellular inflammation, the only inflammatory products are connective-tissue cells. The most frequent examples of this are found in the pia mater and the peritoneum (Fig. 41).

In chronic productive inflammation, the inflammatory product is new connective tissue, with an excess either of basement substance or of cells. The new tissue thus produced may subsequently undergo fatty degeneration or become calcified.

In *connective tissue*, the inflammation produces thickenings and adhesions.

In *mucous membranes*, the growth of new tissue is in the stroma. This is thickened, either diffusely or in the form of polypoid growths. The layer of epithelium may be thickened or thinned. The mucous glands are atrophied, or hypertrophied, or become cystic. The production of mucus is increased or diminished. Such a productive inflammation of the mucous membranes is commonly called a "chronic catarrhal inflammation."

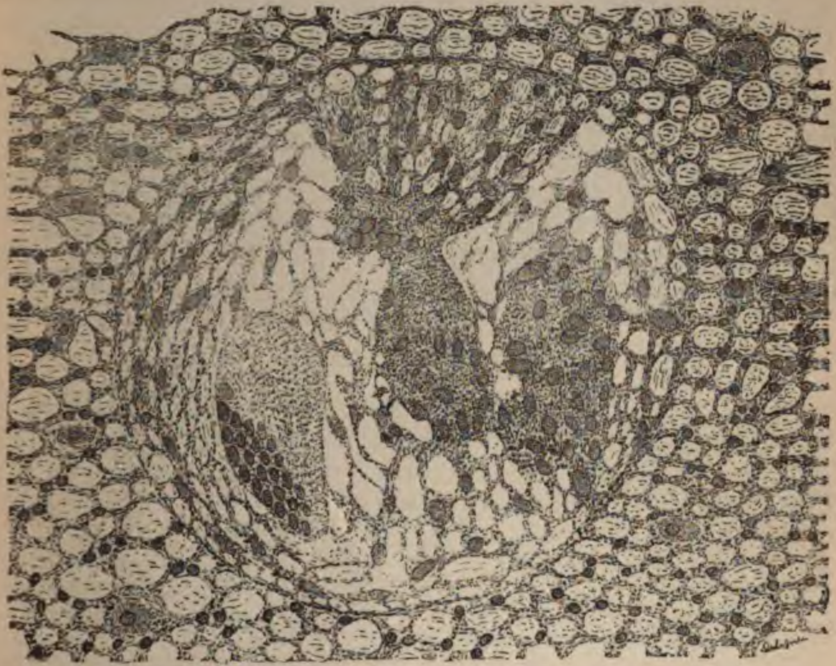

FIG. 42.—A TUBERCLE GRANULUM FROM THE PLEURA, × 850 and reduced. The giant cells form part of the basement substance.

In the *viscera* there is a growth of indifferent tissue or of connective tissue in the stroma. The visceral cells are compressed, or degenerated, or atrophied. The functions of the viscus are seriously interfered with.

The most marked features of this form of inflammation are its slow course and its tendency to continue.

III. TUBERCULAR INFLAMMATION.

Tubercular inflammation is an inflammation accompanying the presence and growth of the tubercle bacillus (see Tuberculosis). The

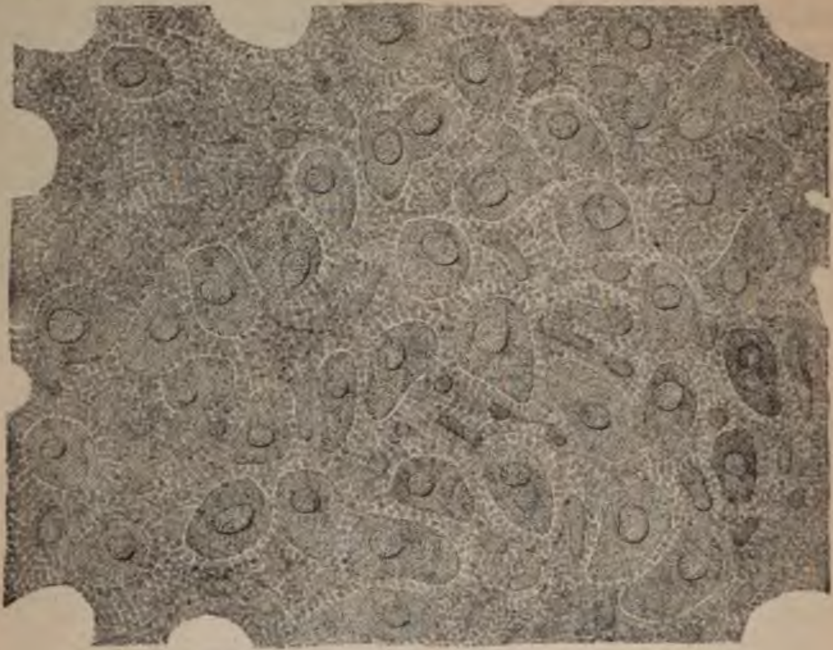

FIG. 43.—DIFFUSE TUBERCLE TISSUE FROM ACUTE PHTHISIS, × 1500 and reduced.

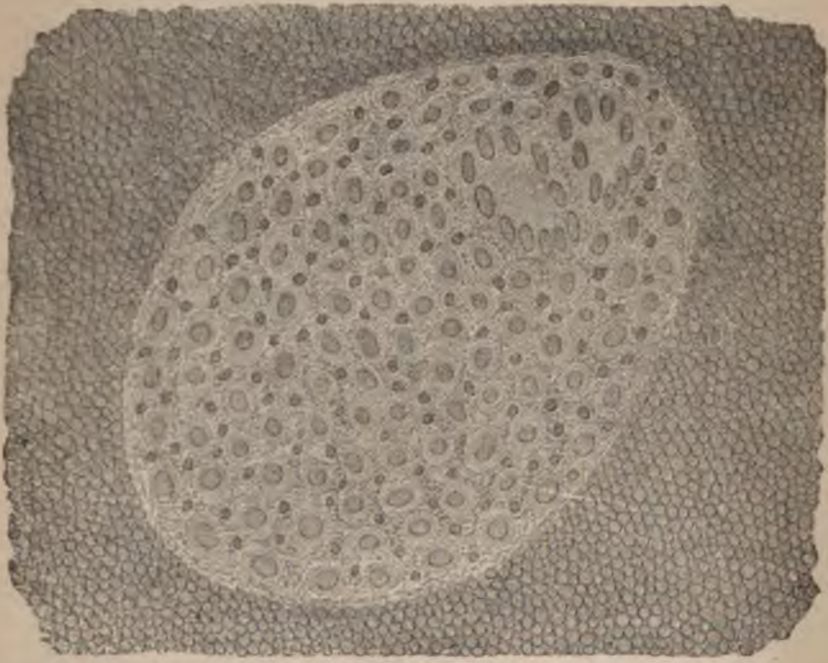

FIG. 44.—A MILIARY TUBERCLE FROM A LYMPHATIC GLAND, × 850 and reduced. The giant cells are inclosed by the basement substance.

inflammation itself is of the exudative or productive type, or of both combined.

The exudative form of inflammation is accompanied with the production of serum, fibrin, and pus.

The productive form is accompanied with the production of round-celled tissue, of fibrous tissue, or of tubercle tissue (Fig. 42). Tubercle tissue is composed of a basement substance forming a meshwork inclosing large polygonal cells, and of giant cells. It is arranged in the form of little spherical bodies—tubercle granula—or of diffuse infiltrations (Fig. 43).

With tubercular inflammations there is often associated obliterating endarteritis. The new tissue produced is of low vitality, so we find that tubercular inflammations are regularly attended with necrosis and cheesy degeneration.

There is also a well-marked disposition on the part of tubercular inflammation to continue and to become chronic.

Tubercular inflammation may be diffuse, involving large areas of tissue; or it may be circumscribed, involving a number of small areas. In the latter case, the little foci composed of products of inflammation are called miliary tubercles (Fig. 44).

IV. SYPHILITIC INFLAMMATION.

Syphilitic inflammation is an inflammation caused by the poison of syphilis, and very probably accompanied by the growth of some specific micro-organism, the nature of which we do not yet know. The inflammation is of the exudative or productive type, or of both combined.

The new tissue is either connective tissue, or small round-celled tissue, or granulation tissue. The connective tissue is especially common in the viscera, taking the form of an interstitial inflammation. The small round-celled tissue and granulation tissue are produced in connective tissue, in the mucous membranes, and in the viscera, usually in the form of circumscribed masses called "gummy tumors," or, in the mucous membranes, "mucous patches." These new tissues possess but few blood-vessels. The vessels in the surrounding tissue are changed by endarteritis. Coagulation necrosis and degeneration soon begin to involve the new tissue.

ACUTE DEGENERATION.

The introduction into the body of certain poisons, such as arsenic, phosphorus, and mercury, and of the poisons of the infectious diseases, is regularly followed by acute degeneration or death of the cells of the viscera.

A small quantity of one of these poisons produces simply a swelling of the visceral cells.

A larger quantity not only causes the cells to become swollen, but they are also infiltrated with granules of albuminoid matter and of fat, and are prone to disintegrate and break down. To these changes may be added a secondary exudative inflammation.

In the more intense forms of poisoning, there is a rapid death of the visceral cells. They either disintegrate and break down, or pass into the condition of coagulation necrosis. With such a rapid death of the cells, a well-marked secondary exudative inflammation is often present.

Some of the examples of such acute degeneration are often called by the name of "parenchymatous inflammation."

TUMORS.

SECTION I. GENERAL CHARACTERS.

Tumors are composed of the same types of tissue as those normally existing in the body, and from the latter they are derived by a proliferation of pre-existing cells. The tissues of tumors may be similar to those of the part in which they grow, when they are called *homologous;* or they may be dissimilar, and are then called *heterologous.* Tumors are not only analogous to the normal tissues of the body in structure, but their life history transpires under the same general laws of nutrition, growth, reproduction, etc. With this important difference, however: that while the normal tissues, serving as they do a definite purpose in the organism, are closely limited in their growth and minute characters by physical and other conditions which determine the uniform development and correlation of various parts, the tissues of tumors exhibit a certain lawlessness in growth, structure, and life history which gives them a distinctive character while not removing them from the physiological types. Thus in the Chondromata,[1] the tissue, while distinctly cartilaginous in type, presents itself not only in places where it does not belong, but may show a tendency to the development of fibres in one part of its basement substance, while another may be distinctly hyaline, or another soft and almost gelatinous. The cells also are apt to exhibit great lack of uniformity in size, shape, and grouping. The lawlessness in tumor tissues is shown in their tendency, under certain conditions, to change from one form into another, as from fibrous tissue into bone.

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Tumors are supplied with blood-vessels which grow into them from adjacent healthy parts, just as they do into granulation tissue, so that they may finally possess a more or less independent vascular system of arteries, capillaries, and veins. They are furnished with lymph vessels and some of them with nerves. The cell division by which tumors grow exhibits the same minute phenomena as does cell division in normal tissues. Tumor tissues are subject to the same degenerative changes as other tissues; they may become fatty or calcified, ulcerated, gangrenous, pigmented, etc. By necrotic changes a tumor may be largely destroyed, but complete obliteration rarely occurs in this way. They are liable to undergo the ordinary inflammatory changes, granulation tissue may form in them, and abscesses and cicatrices.

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[1] Tumors are designated by the termination *oma* (plural *omata*).

The rapidity of growth of tumors varies greatly; some grow very slowly indeed, and may change but imperceptibly in size and appearance for years, while others, on the other hand, grow so fast that they do not acquire solidity, and their elements remain in an incompletely developed condition, and are thus more liable to destructive changes than normal tissues are. In healthy tissues, the blood-vessels are supported by surrounding elements, which aid them in sustaining the blood-pressure from within. In rapidly growing tumors, this external support is often lacking, and, as the walls of the blood-vessels are themselves badly formed, the result is that the walls are apt to become pouched or aneurismal, and they often burst, giving rise to larger or smaller interstitial hæmorrhages.

Tumors have various shapes: nodular, tuberous, fungoid, polypoid, papillary, dendritic, etc.

Tumors may occur singly or in greater or less numbers in the same or in different parts of the body. If they are multiple, they may have occurred simultaneously or at different times as independent structures. Or multiple tumors may occur as the result of the dissemination in the body, from a primary tumor, of cells which form a starting-point for new tumors. Many tumors are sharply circumscribed, may be even incapsulated, and influence surrounding parts only by the pressure which they exert upon them. In this way they may cause displacement, atrophy, or necrosis; they may, by pressure on neighboring vessels, cause œdema, thrombosis, etc.; they may in the same way cause dislocation and caries of bones.

Tumors may grow largely by increase of elements within them, thus simply expanding; this is called *central growth*. They may grow in part or largely at the surface—*peripheral growth*. In this case the growth may be a direct, continuous enlargement of the mass at or near the periphery, or it may be by the formation of secondary nodules near the primary growth, which, gradually enlarging, finally coalesce with the latter, forming a part of the nodular tumor. This mode of enlargement is called *discontinuous peripheral growth*, and is due to the dissemination of cells from the mother tumor into the adjacent tissue, through the blood or lymph channels, and their proliferation at the points of lodgment. This dissemination may occur by the agency of blood or lymph currents or by the amœboid movements of the cells.

It is not yet certain whether the new cells which are produced in tumors are altogether the result of the proliferation of the primary tumor cells, or whether the ordinary tissue cells of the part, connective-tissue cells, white blood-cells, etc., may undergo transformation and proliferation under the influence of the characteristic cells of the tumor. It is not unlikely that both modes of increase occur, although the former is probably the more common and important. Some tumors increase by an infiltration of surrounding tissues, whose elements they gradually replace. In certain tumors, the old tissue of the part in which they grow may remain with its vessels, and form a sort of matrix whose interstices are infiltrated with the new tumor tissue. The irritation of the tumor may induce inflammatory new formation of tissue of the old matrix about or within the tumor.

But all tumors are not limited to that part or region of the body in which they first occur. Sooner or later, secondary nodules resembling the first may be found in the most distant parts of the body, sometimes singly, sometimes in great numbers. These may grow like the parent tumor, and themselves form foci for new disseminations.

This dissemination of tumors is one of the most important elements of malignancy, and is called *metastasis*, the secondary tumors being called *metastatic tumors*. This occurs by the transportation of tumor cells through the blood or lymph channels. Since the tumor itself may be filled with new and badly formed blood and lymph vessels, and its structures be in close contact with the vessels of the tissue in which it grows, the cells of the primary tumor may, by ulceration through, or by atrophy of, the walls, readily find their way into the lumen of the vessels, and be swept away by currents as emboli, and, finding lodgment, proliferate and grow, forming secondary tumors; or the proliferation may occur in the vascular endothelium itself, when the formation of emboli is easy to understand. When carried through the lymph vessels, the tumor cells may for some time be kept from the larger channels and from general dissemination by lodgment in the lymph glands, where they may establish independent tumors. The parts of the body in which metastatic tumors are most apt to form depend, of course, upon the situation of the primary tumor and the distribution of the vascular channels through which dissemination occurs.

The tumors in which metastasis is most apt to occur are, as a rule, those which grow rapidly, are vascular and succulent, and contain many cells.

Not less variable than the size, mode of growth, and structure of tumors is their significance in the organism. Surgeons have in the past, and to a certain extent still do classify tumors, for practical purposes, as *malignant* and *benign*, and for a long time malignant tumor and cancer were synonymous terms. Now we know that other tumors as well as cancers are malignant, and, furthermore, contrary to the former belief, that malignancy does not depend upon any specific agent in the tumor. If we mean by a malignant tumor one which may cause death, any tumor may be malignant if growing in the right place. Thus a simple fat tumor, by pressing on the trachea, may cause suffocation, and any tumor may secondarily cause death by hæmorrhage or septicæmia. The real signs of malignancy in a tumor are: 1. Invasion of adjacent tissues by eccentric or peripheral growth. 2. The tendency to local recurrence after removal. 3. The formation of metastases. 4. A tendency to interfere with the nutrition and general well-being of the body, which may give rise to a condition known as cachexia. The modes of invasion of surrounding tissues and the formation of metastases have been considered above. The tendency to local recurrence after removal is probably, in most if not all cases, due to the incomplete removal of the peripheral infiltrating cells. These may be very few in number and lacking in characteristic structural features, but are none the less endowed with the capacity of proliferation and development into a new and similar tumor at or near the seat of the extirpated one. The infiltrating peripheral cells may remain dormant for a long time after an operation, or may immediately commence to grow. The mere fact that a second tumor develops in the place of one removed does not imply malignancy, since it may result from the same mechanical cause which produced the first, as in the case of certain carcinomata of the lip induced by the mechanical irritation of a pipe.

The drain upon the system by the rapid growth of a tumor, together with the absorption from it into the body of deleterious putrefactive materials, from sloughing, ulceration, and degeneration, may give rise to fever and other constitutional disturbances. Or they may induce feebleness, anæmia, and that general impairment of the nutritive functions of the body known as cachexia. This condition is frequently rendered worse by the mental status of the patient in the presence of such a traditional object of alarm.

It should be remembered, however, that so long as they are localized and have not undergone degenerative changes, even the most malignant tumors do not usually give rise to a cachexia, since the drain upon the nutritive powers of the system by their simple growth is not, under ordinary conditions, very considerable. When the system is deteriorated by the absorption of septic materials from tissue degeneration, however, this may become a very important factor.

This condition of cachexia, so evidently secondary to the growth and degeneration of the tumor, was formerly termed a dyscrasia or diathesis, and was supposed to precede and induce the growth of malignant tumors, particularly cancers.

It is further to be noted that the fragments of tumors which have found access to the veins may act as simple emboli and produce immediate death, or simple metastatic abscesses.

THE CAUSE OF TUMORS.

In regard to the causation of tumors, our actual knowledge is still very meagre. In a certain number of cases, mechanical influences are undoubtedly sufficient inciting causes. In other instances, heredity is an important factor. But to both of these influences too much importance has been attributed in former times. The most recent, and to a certain extent plausible, hypothesis, and one which most satisfactorily accounts for the occurrence and character of many tumors, is that of Cohnheim, called the *hypothesis of the embryonal origin of tumors.* This is to the effect that all true tumors are due to faulty embryonal development; that certain embryonal cells of various kinds, in the course of the development of the body, are superfluous, or become displaced, or do not undergo the normal changes, and remain ready, when the conditions shall become favorable in later life, from whatever reason, to commence growing with all the potencies of embryonic and lowly organized cells in the midst of the mature tissues. Not being restrained, however, by the regulating influences which determine the nature and relative extent of growth in normal development, they go on to the production of tumors, which represent, though in atypical form, the various tissues which the strayed or unused cells were destined normally to produce.

The evident hereditary character of many tumors, the congenital nature and early development of others, their atypical structure in general, and the tendency of many forms to occur in situations in which, during the development of the embryo, considerable complexity exists, as well as their heterologous occurrence and their frequent primary multiplicity—all of these characters of tumors seem to favor Cohnheim's hypothesis. On the other hand, the theory leaves unexplained the sudden growth of the alleged germs which have long remained dormant, and lacks as yet the absolute demonstration of a morphological basis, since no one has seen the strayed or delayed embryonic cells. These may, of course, be very small and difficult of demonstration, and this, according to Cohnheim, fully explains the lack of a definite histological basis to his hypothesis. It should be remembered, furthermore, that, under ordinary conditions in the body, certain cells which are destined to replace others which have fulfilled their destinies, as in the skin, possess to a greater or less degree the characters of embryonal cells, and that, while in the struggle for existence the growth of these cells may be held in check, as by conditions of pressure, nutritive supply, etc., if these conditions be altered these cells may undergo proliferative changes as significant as those of the alleged belated germs of Cohnheim. Such a changed condition of affairs has been shown by Thiersch to occur frequently in the skin in old age, and to explain in large measure the occurrence of certain epithelial tumors.

It should be remembered that this hypothesis was offered by Cohnheim only as a suggestion to facilitate research, and that he expressly warned his confrères against attaching a premature importance to the possibility to which he called attention. Thus, while the hypothesis of the embryonal origin of tumors is most fascinating, and for certain forms quite satisfactory, we may well demand a more definite basis of fact before accepting it as of universal application.

It was formerly supposed, when the doctrine of the specific nature of tumors prevailed, that the cells of malignant tumors, particularly of cancer, had a characteristic structure and appearance, and that by the examination of single or of a few separated cells the nature of the tumor could be determined. From the above considerations, it will be evident, as all tumor cells have their prototypes in the normal body, that therefore there is nothing pathognomonic in the appearances of single cells. It is by a study of the general structure and of the topography of tumors, as well as of the characters of the individual cells, that we are enabled to determine their nature. And even then we must often bring to our aid the clinical history and gross appearances of the growth before we can arrive at a definite conclusion. We may, indeed, sometimes, aided by the clinical history or gross appearances, be able, by the microscopical examination of scrapings from a tumor or of fluids from an internal cavity in which it is growing, to form a reasonable conjecture regarding its nature.

As a rule, the peripheral portions of the more rapidly growing tumors are best adapted for microscopical examination, because here secondary degenerative changes are less likely to have occurred than in the central parts.

Bacteria have in recent times been claimed by some observers to stand in a causative relation to certain tumors, and bacteria have been occasionally demonstrated in, and cultivated from, the tissues of tumors. But no complete and reliable experiments or observations have as yet been made which prove that they have anything to do in causing the tumors, or are of any significance save as chance contaminations of the tissues or as inducing secondary complications.

CLASSIFICATION OF TUMORS.

The fact that tumors are composed of structures which resemble the various types of tissue found in the normal body suggests the guiding principle in their classification. But in order to thoroughly understand either the classification of normal tissues or the grouping of the tumors, we must keep in mind the way in which the tissues are developed in the embryo.

According to the more recent views of embryologists, particularly of His and Waldeyer, the primitive tissues of the body belong to two groups: those of *archiblastic* and those of *parablastic* origin. In the early stages of fœtal development, the new cells which are formed at first arrange themselves in three layers, to which collectively the name *archiblast* is applied.

Of these three archiblastic layers, the outer, called the *epiblast*, furnishes the material for the epithelium of the skin and its adnexa, for the epithelium of the terminal portions of the alimentary canal, and for the nervous system, including the neuroglia.

The middle layer—the *mesoblast*—furnishes the material for the epithelium of the genito-urinary organs, and for both the smooth and striated muscle tissue.

The inner layer—the *hypoblast*—affords the material for the development of the epithelium of the respiratory and the digestive systems, with that of the various glands and passages which develop out of and in connection with them.

The exact origin of the *parablast*, which develops later than the archiblast, is still uncertain; but it furnishes the material out of which are formed the connective tissues, including cartilage, bone, teeth, and fat; the blood-cells and blood-vessels; the lymphatic tissues and lymph vessels, and the true endothelial cells.

Now, if we wish to arrange in groups the different kinds of tumors found in the body, we have only to recall the varieties of tissue which normally exist there, and their grouping, and upon the classification of the physiological types to construct the classification of tumors. It should be remembered that the usual separation of the normal tissues into groups is useful, rather because it facilitates their study than because it expresses absolute and fundamental distinctions; and the same may be said of all the classifications of tumors. In both, an increase of our knowledge concerning their structure and genesis will doubtless lead to a more accurate grouping; but, for the present, such an arrangement as that indicated below will be found of practical value for the purposes of studying tumors.

I. Tumors Composed of Tissues of the Type of those forming the Connective-Tissue Group.—Histioid or Connective-Tissue Tumors.

Physiological Type.	*Tumors.*
1. **Fibrillar connective tissue.**	1. Fibroma.
2. **Mucous tissue.**	2. Myxoma.
3. **Embryonal connective tissue.**	3. Sarcoma.
4. **Endothelial cells.**	4. Endothelioma.
5. **Fat tissue.**	5. Lipoma.
6. **Cartilage.**	6. Chondroma.
7. **Bone.**	7. Osteoma.
8. **Neuroglia.**[1]	8. Glioma.

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[1] It will be seen, from the account given above of the origin of the various tissues in the different embryonic layers, that the neuroglia has a different origin from the other

II. Tumors Composed of Tissues of the Type of Muscle Tissue.—Myomata.

Physiological Type.	*Tumors.*
1. Smooth muscle tissue.	1. Leiomyoma.
2. Striated muscle tissue.	2. Rhabdomyoma.

III. Tumors Composed of Nerve Tissue.—Neuromata.

Physiological Type.	*Tumors.*
1. Nerve tissue.	1. Neuroma.

IV. Tumors Composed of Vascular Tissue.—Angiomata.

Physiological Type.	*Tumors.*
1. Blood-vessels.	1. Angioma.
2. Lymph-vessels.	2. Lymphangioma.

V. Tumors in which the Predominant or Characteristic Elements are Epithelial Cells.

Physiological Type.	*Tumors.*
1. Glands.	1. Adenoma.
2. Various forms of epithelial cells and associated tissues.	2. Carcinoma.

VI. Tumors Formed by Various Combinations of the above Types.—Mixed Tumors.

Aside from the above well-marked classes, we may mention here for the sake of completeness:

(*a*) *Complex Congenital Tumors—Teratoma.*—These are congenital tumors which frequently contain a great number of different forms of tissue, such as various forms of fibrillar connective tissue, cartilage, bone, teeth, hair, skin, muscle, and glands. They are most frequently found at the lower end of the spine, about the head and neck, or in the generative organs. Some of them probably arise by an inclusion of portions of another fœtus. These are called *teratoid tumors*, or *teratomata.* Among them are sometimes classed other and simpler congenital formations, such as dermoid cysts, congenital angiomata, and the so-called pigmented moles.

(*b*) *Cysts.*—These structures, for the sake of convenience, are usually classed among the true tumors, although in general characters, structure, and genesis they are entirely different products. They are usually divided into two classes:

I. *Cysts which develop in pre-existing cavities.*

II. *Cysts which originate independently as the result of pathological changes.*

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connective tissues. The neuroglia, as well as the tumors derived from it, presents marked peculiarities in structure, but its structural and functional alliance with the other connective tissues justifies its grouping among them.

I. Cysts which develop in pre-existing cavities:

1. *Retention Cysts.*—These are chiefly formed by the accumulation in glands or their excretory ducts of the more or less altered secretion of the gland. They usually occur as the result of some hindrance to the normal discharge, as from inflammatory contractions, pressure, etc. The contents of such cysts are usually mucous, sebaceous, serous, or of a mixed character. Their walls are the more or less altered walls of the original structure. To this class belong comedones, milium, atheroma, chalazion, ranula, the ovula Nabothi, milk cysts, and certain serous cysts of the ovaries, Fallopian tubes, gall-ducts, and uriniferous tubules.

2. *Exudation Cysts.*—These arise usually, though not always, as the result of a chronic inflammatory process in lymph spaces or serous sacs, and among them are to be classed the so-called ganglia, hydrocele, etc. Certain of the so-called hæmatoceles, in which blood is extravasated into closed cavities, form a variety of the cysts of this group.

II. Cysts which originate independently as the result of pathological changes:

1. *Cysts formed by the softening and disintegration of tissue.*—Such cysts may at first be small and have very meagre contents and no well-defined wall. A wall may finally be present either as an entirely new-formed structure, or the more or less modified capsule of the organ in which they occur may partly or entirely form the wall. The contents of such cysts are usually the more or less altered detritus of the tissue by whose disintegration they are formed. Such cysts are very apt to occur within true tumors, particularly those which are succulent and of rapid growth, since these, as above stated, are very liable to degeneration. Old abscesses may change into well-defined cysts of this kind.

2. *Cysts formed around foreign bodies.*—The inflammatory reaction induced by the presence of foreign bodies of various kinds, parasites, masses of extravasated blood, etc., frequently result in the formation of well-defined incapsulated cysts.

3. *Cysts formed by a new growth of tissue in whose spaces various kinds of fluid accumulate.*—These spaces may or may not be lined with epithelium and have something of the glandular character. Such forms are exemplified in some of the compound ovarian cysts—the so-called ovarian cystomata.

4. *Congenital Cysts.*—These are of various forms, and their mode of origin is in most cases but imperfectly understood. The so-called dermoid cysts of the subcutaneous tissue and ovary are marked examples of this class. Certain cougenital cysts of the kidney and other internal organs are conveniently grouped in this class, although it is quite probable that some of them at least originate during fœtal life in one or other of the above-described ways, and hence are not essentially different in nature from some of the cysts of other classes.

Various Lesions sometimes described as Tumors.—There are certain enlargements of the lymph glands or nodes which are in reality hyperplasias, sometimes inflammatory in character, and sometimes not, and which are often grouped among the tumors as *lymphomata*. They are not, strictly speaking, true tumors, and will be considered under the lesions of the lymph nodes.

In the same group are often classed the enlargements of the lymph glands in leukæmia and in other general diseases, which will be treated in another part of this book. Another group of tumors, sometimes called lymphomata, are in reality sarcomata, and these will be described under the latter heading.

There is also a group of nodular new formations, the so-called *Infective Granulomata*, which are sometimes classed among the tumors. These are found in tuberculosis, leprosy, syphilis, lupus, glanders, and actinomycosis. They seem, however, to be more closely allied to inflammatory new formations than to true tumors, and, as our knowledge regarding them increases, seem more and more to be dependent upon the irritation caused by the presence of vegetable parasites (see section devoted to General Diseases).

Nomenclature of Complex Tumors —The simple occurrence of more than one kind of tissue in a tumor does not make it a complex or mixed tumor. It is only when a special kind of tissue occurs in sufficient quantity to be of definite significance, or is of such a nature as to render its presence, in any amount, of importance, that we recognize its presence in the name. The name of mixed tumors is usually formed by joining the names of the tissues to be recognized. Thus a combination of fibroma and sarcoma is called fibro-sarcoma; the general rule of construction being that the name of the more important tissue shall serve as the substantive which that of the less important one qualifies. It should be remembered, however, that the more important tissue is not always the one which is present in greatest amount. Thus, owing to the great clinical significance of carcinomatous tissue, a very large fibroma with a small quantity of cancer tissue intermingled would be a *fibro carcinoma* and not a carcino-fibroma.

Preservation.—In general, tumors, like all tissues for microscopical study, should be cut into small pieces before immersing them in the preservative fluids, and the sooner they can be placed in these after removal, the better will be the preservation. In some cases, much may be learned from large sections of tumors together with their surrounding tissues. In this case, the proper part of the tumor must be preserved whole, and is best hardened in strong alcohol. For the ordinary routine hardening of tumors, Müller's fluid is probably the best agent; the hardening being completed in the usual way with alcohol. In many cases an interstitial injection of one-per-cent aqueous solution of osmic acid, or a mixture of this with equal parts of alcohol and water, and subsequent immersion in alcohol, secures a very perfect preservation of the cells.

When it is desired to study the living cells of tumors—and much may be learned in this way—fragments should be teased in one-half-per-cent salt solution and examined on a warm stage. For purposes of immediate diagnosis, sections may be made of the frozen tissue and stained with safranin.

SECTION II. SPECIAL FORMS OF TUMORS.

FIBROMA.

The fibromata are composed of fibrillar connective tissue, which, as in the physiological type, is sometimes dense and firm, *Fibroma durum,* and sometimes loose in texture and soft, *Fibroma molle.* They are usually sharply circumscribed and are frequently incapsulated, but they may be diffuse and merge imperceptibly into the surrounding tissue. Some fibromata consist almost entirely of intercellular substance, containing but few flattened or spindle-shaped cells (Fig. 45); others contain very many variously shaped cells. The denser varieties usually contain but few blood-vessels, although they are occasionally quite vascular. Many of the softer varieties are very vascular. Nerves also are occasionally seen. The course and arrangement of the fibres in these tumors are usually quite irregular, often crossing and interlacing in a most complex manner. The fibromata are usually of slow growth, but exceptionally they grow very rapidly. They are benign tumors, but by pressure on important organs, by ulceration, or by changing into other varieties of tissue, they may become of serious import. Pure fibromata do not form metastases, but they are often multiple, and when so are frequently congenital. They may, like most tumors, exhibit local recurrence when not fully removed. They are frequently very small and insignificant, but, on the other hand, may grow to an enormous size.

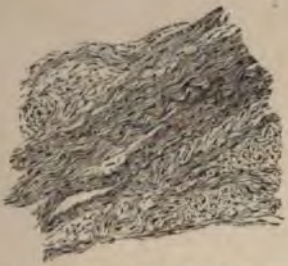

FIG. 45.—DENSE FIBROMA OF THE ABDOMINAL WALL.
Some of the bands of connective-tissue fibres are cut across, others are cut lengthwise.

They are quite frequently combined with other kinds of tissue to form complex tumors. The looser, softer varieties not infrequently become œdematous, when they may closely resemble myxomata. They are liable to calcification, and to fatty and mucous degeneration. By metaplasia they may partially change to form fibro-chondroma, fibro-lipoma, fibro-sarcoma, or fibro-osteoma. The latter transformation frequently occurs when they form in the periosteum. Developing as they do in the connective tissue, they occur in the most various parts of the body: in the skin and subcutaneous tissue; in intermuscular tissue and fasciæ; in periosteum; in the nerve sheaths and intrafascicular connective tissue; in the dura mater, the interstitial tissue of organs, and in the mucous membranes. Many of the so-called polypi of the mucous membranes, some psammomata, certain forms of warts and common papillomata, are forms of fibroma.

Occasionally, in the ducts of glands, fibrous polypi grow to an enormous extent, their epithelial covering keeping pace in growth with their development, until they form very large, irregular, loose-textured tumors, which often finally ulcerate. Such forms are seen in the mammary gland, where they are frequently mistaken for carcinomata. They are called *Intracanalicular Fibromata* (see Tumors of the Mamma). It is often difficult to distinguish between genuine fibromata and inflammatory or other connective-tissue hyperplasias, such as elephantiasis, and perhaps the fuller knowledge of the future will show that the distinctions are not as definite as we are now disposed to believe.

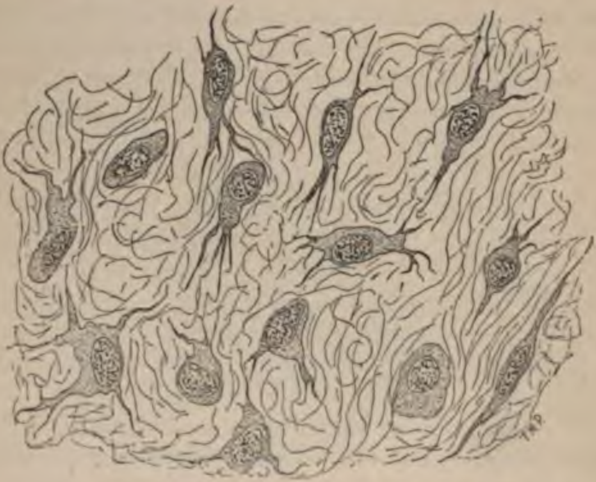

FIG. 46.—MYXOMA OF SUBCUTANEOUS TISSUE OF BACK.

MYXOMA.

Mucous tissue is essentially an embryonic tissue, for in the normal adult it is present only in a very imperfect and atypical form in the vitreous of the eye, and perhaps exceptionally in small amount about the heart, kidneys, and medulla of bone.

The myxomata are thus essentially embryonic-tissue tumors. These tumors consist, in their most typical forms, of a homogeneous or finely fibrillated, soft, gelatinous basement substance, in which are imbedded a variable number of spheroidal, fusiform, branching, and often anastomosing cells (Fig. 46). They may contain few or many blood-vessels and nerves. By the addition of acetic acid, mucin may be precipitated from the basement substance. The very soft forms which contain comparatively few cells and much translucent basement substance are called *Myxoma gelatinosum* or *M. molle.* The presence of many cells renders them more consistent and gives them a whiter and more opaque appearance; such forms are called *M. medullare.*

Pure myxomata are not very common. The myxomata are very apt to be combined with fibrillar connective tissue as *fibro-myxoma;* or with fat tissue—*lipo-myxoma;* and they very frequently become sarcomatous, or take part in the formation of very complex tumors. They may be diffuse or incapsulated with fibrillar connective tissue; they are frequently very large, and may be multiple. Owing to the character of the basement substance, the blood-vessels not infrequently rupture, giving rise to larger or smaller hæmorrhages within the tumor, or to the formation of cysts. The cells are liable to undergo fatty degeneration (Fig. 47).

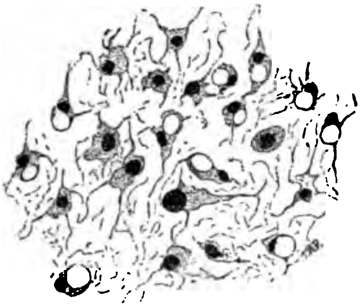

FIG. 47.—MYXOMA GROWING INTO ABDOMINAL CAVITY.
Showing the accumulation of fat droplets in some of the cell bodies.

Composed as they are of a type of tissue from which fat tissue is developed in the embryo, the relations of these tumors to fat tissue are very intimate. They are most frequently developed in, and probably directly from, fat tissue, and are very often combined with it as lipo-myxoma. They are also found in the subcutaneous, submucous, and subserous tissue, in the marrow and periosteum; in the brain and cord; in the sheaths and intrafascicular tissue of peripheral nerves; in intermuscular septa, and in the interstitial tissue of glands, such as the mamma and parotid. The myxomata are in general benign; yet they are very prone, especially the lipomatous forms, to local recurrence. They som times grow very rapidly, and sometimes, though very rarely, form m tastases. In the not infrequent combination with sarcoma, they may exhibit the most marked malignancy. Many of the polypi of mucous membranes are myxomata, and to this class of growths belong the so-called hydatid moles which sometimes develop in the villi of the chorion.

Œdematous, loose, and cellular forms of fibrillar connective tissue so closely resemble some of the forms of mucous tissue that certain observers consider them as identical. So prone are many tumors to undergo mucous degeneration, and so frequent are the combinations of the myxomata with other forms of tumors, that it is often difficult, sometimes impossible, to say whether the mucous tissue in a given composite tumor is primary or secondary.

SARCOMA.

These tumors are formed on the type of connective tissue, but they are, as a rule, largely composed of cells; the basement substance, though a constant and important factor, being much less conspicuous than in adult connective tissue. They more closely resemble, in general, the developing connective tissue of the embryo or the granulation tissue of inflammation. They are, therefore, conveniently described as presenting the type of embryonal tissue. The prominent and characteristic cells of the sarcomata are most varied in size and shape. They may be fusiform or spindle-shaped, spheroidal, branched; they may be multinuclear and very large, or they may be very small and spheroidal, resembling leucocytes. The fibrillar basement substance may be present in such small quantity as to entirely escape a superficial observation, covered as it may be by the abundant cells; or it may be so abundant as to give the tumor the general appearance of a fibroma. It may be intimately intermingled with the cells in fascicles, or it may be in large open-meshed networks, giving to the tumor an alveolar appearance. The cells, however, always stand in an intimate relationship to the basement substance, which they sometimes reveal by fibrillar processes continuous with it. Blood-vessels also form a constant and important structural element in these tumors, being in some of them so predominating a factor that they give structural outline and general character to the growth. They, too, as in the normal connective tissue, are intimately associated with the basement substance and with the tumor cells.

A single form of cells is often so predominant as to furnish a suitable name for the tumor, but in many cases the cell form varies greatly in the same growth. It may be said in general that there is a tendency to reproduce in these tumors some of the special characteristics of the tissues in which they originate. Thus, sarcomata of the bones are apt to be osteo-sarcomata; those of pigmented tissue, like the choroid, are apt to be pigmented sarcomata. It will be more convenient for our present purpose to briefly describe the more common forms one after another than to attempt any systematic classification of them.

It should be remembered, however, that the various forms are not sharply specific in character, but are apt to merge into one another and to intermingle in various ways.

Sarcomata are most frequently found in the skin, subcutaneous tissue, fasciæ, subserous connective tissue, the marrow or periosteum, and in the choroid. They may also occur, though more rarely, in the dura mater; brain and cord; lymph nodes; in the adventitia of blood-vessels, and in nerve sheaths; in submucous tissue; in the uterus, and in the kidney. In the liver and lungs and heart they may occur by metastasis.

They are more apt to occur at an early period in life than later. The cellular character, the rapid growth, the vascularity and succulence of many forms, the marked tendency to local recurrence, and the formation of metastases, stamp the sarcomata as malignant tumors. But in this they vary greatly; while some of the forms belong in every sense to the most malignant of tumors, others grow slowly, are very dense, and may remain localized and harmless for years. Their tendencies in this respect will be mentioned under the special forms.

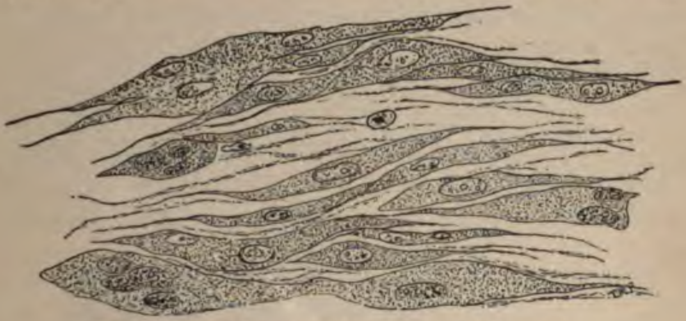

FIG. 48.—LARGE SPINDLE-CELLED SARCOMA OF HUMERUS.

Intimately related as they are to the blood-vessels, metastasis is more apt to occur through the blood than through the lymph channels, and consequently adjacent lymph glands are much less apt to be involved than in some other forms of tumor, notably the carcinomata.

Spindle-celled Sarcoma.—The cells in these tumors may be large—*large spindle-celled S.* (Fig. 48); or they may be small—*small spindle-celled S.* (Fig. 49). They may consist largely of cells, or may contain so much intercellular fibrous tissue as to be appropriately called *fibrosarcoma.* The cells are frequently arranged in fascicles, which surround the blood-vessels, and these fascicles may cross and interlace. These tumors are, as a rule, denser and firmer and less malignant than other forms of sarcoma, but to this there are many exceptions. They may be incapsulated or infiltrating. To this class belong the growths formerly described as fibro-plastic tumors and recurrent fibroids. They frequently occur in the periosteum, subcutaneous tissue and muscle; in the uterus, and in various glands, notably in the mamma, testicle, thyroid, etc. These forms are among the most frequent of the sarcomata. .

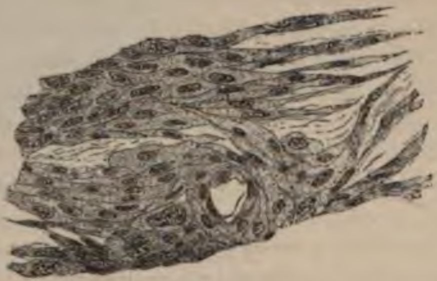

FIG. 49.—SMALL SPINDLE-CELLED SARCOMA OF FOREARM.

Round-celled Sarcoma.—Of these there are two classes—1, *small round-celled sarcomata*, and, 2, *large round-celled sarcomata.*

1. The small round-celled sarcomata consist of cells of about the size and appearance of lymph cells, and may have much or little intercellular substance, which may be irregularly disposed or arranged in large meshes resembling alveoli. In many cases, so small is the quantity of intercellular substance that it is difficult of detection without special modes of preparation. These tumors are apt to contain many blood-vessels, and be very soft and succulent (Fig 50). Their growth is sometimes rapid and they are often very malignant.

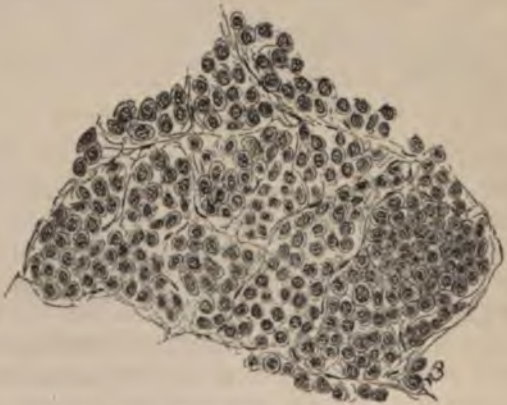

FIG. 50.—SMALL ROUND-CELLED SARCOMA OF LIVER.

They most frequently occur in the connective tissue of the muscles and fasciæ, in bone, and in lymph nodes (*lympho-sarcoma*). They also occur in the internal organs, not infrequently in the brain, associated with glioma as *glio-sarcoma.*

2. In the large round-celled sarcomata (Fig. 51), the cells vàry in size, but are usually very much larger than in the last variety. Their nuclei are usually large and contain prominent nucleoli. They, too, are often very vascular, and contain a variable quantity of basement substance. They are occasionally alveolar in character. They are as a rule less soft and malignant than the small-celled varieties.

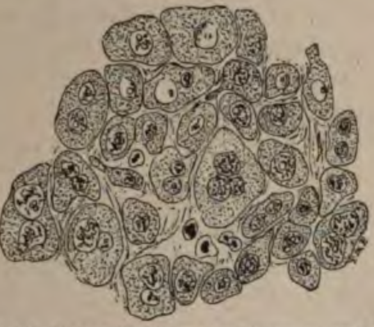

FIG. 51.—LARGE ROUND-CELLED SARCOMA OF LEG.

The round-celled sarcomata were formerly supposed, on account of their macroscopical and clinical resemblance to some of the soft forms of carcinoma, to belong to these tumors, and were called medullary cancers.

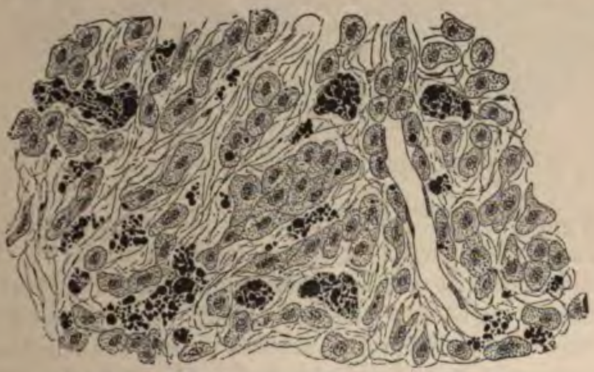

FIG. 52.—MELANO-SARCOMA FROM SUBMAXILLARY REGION.

Melano-Sarcoma.—These tumors consist most frequently of spindle cells of various sizes, although cells of other shapes frequently occur in them. They are characterized by the presence in the cells, and less frequently in the intercellular substance, of larger and smaller particles of brown or black pigment (Fig. 52). The pigment is usually quite irregularly distributed in patches or streaks, and is located chiefly in the cell body. They arise most frequently in the skin and in the choroid. Pigmented moles of the skin often form their starting points. They belong to the most malignant of tumors. They very readily form metastatic tumors in various parts of the body, which are, like the parent tumor, pigmented.

Various forms of tumors may contain brownish pigment deposited in them by the degeneration of the hæmoglobin from extravasated blood; these should not be mistaken for melanotic sarcomata.

Myeloid or Giant-celled Sarcoma.—Tumors of this class are usually formed chiefly of spheroidal or fusiform cells of variable size, but their characteristic feature is the presence of larger and smaller multinuclear cells, called giant cells. These are closely intermingled with the other cells, and may be very abundant or very few in number (Fig. 53). Giant cells may occasionally occur in other tumors, but are most abundant and characteristic in these. They are chiefly formed in connection with bone, and may commence in the marrow or in the periosteum. They are sometimes very soft and vascular, and subject to interstitial hæmorrhages. Some of these vascular sarcomata were formerly classed together with other kinds of vascular tumors as fungus hæmatodes. Some of the forms of *epulis* are giant-celled sarcomata.

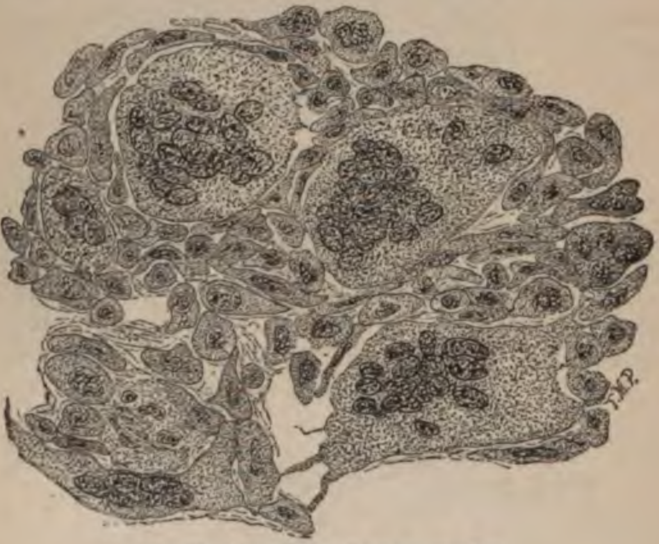

FIG. 53.—GIANT-CELLED SARCOMA OF BONE.

When these tumors originate in the marrow of the long bones, which is a favorite place for them, they are apt to cause resorption of the bone, and although the tumor may be for a long time inclosed by a shell of new-formed bone, which enlarges with the enlarging tumor, it usually, sooner or later, breaks through this and infiltrates adjacent tissues. They are liable to form metastases and frequently grow to a very great size. The periosteal forms are apt to be firmer in texture, and are prone to the development of irregular masses of new bone within them, thus forming one of the varieties of osteo-sarcoma.

Osteo-Sarcoma.—These are spindle or round-celled tumors, usually, but not always, connected with bone, in which irregular masses of bone tissue are present. The bone is usually of irregular atypical structure; the regular lamellation and typical Haversian canals being usually absent. They may form metastases which present similar characters.

Calcification, which should be distinguished from ossification, may occur in various forms of sarcoma.

Angio-Sarcoma.—In many of the sarcomata in various parts of the body, the blood-vessels form so prominent and important a feature as to give special character to the growth, not alone by their size and general prominence, but sometimes by the peculiar arrangement which their presence gives to the cells. While in most of the sarcomata the blood-vessels have a very important influence in determining the topography of the tumor, in most of the denser, and in many of the softer varieties, this influence is not easily traced. In many forms, however, particularly those which are soft and very cellular, the cells are closely grouped around the vessels, as if they were developed in their adventitiæ and had formed close sheaths around them. The masses of cells thus formed, with a blood-vessel for a centre, may be closely packed together in long strings with more or less frequent anastomoses (Fig. 54), or they may be

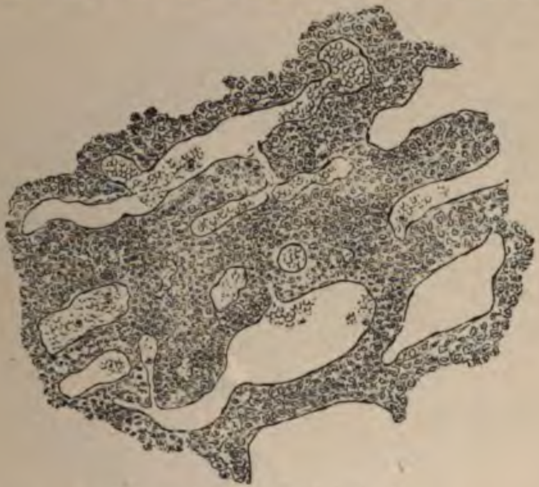

FIG. 54.—ANGIO-SARCOMA OF LIVER.

The thin-walled blood-vessels, around which the tumor cells are grouped, are irregularly dilated.

arranged in rounded groups, giving to the tumor an alveolar appearance.

In other cases, the blood-vessels may appear as characteristic factors simply on account of their size or relative abundance.

Sometimes the walls of the blood-vessels and the adjacent tissues, in these as in other forms of tumors, undergo a peculiar hyaline degeneration, giving to the whole or to parts of the tumor a more or less gelatinous appearance. Such tumors are not very common, and are frequently grouped in an ill-defined class called *cylindroma*.

Alveolar Sarcoma.—Sometimes, as above stated, the basement substance of the sarcomata, particularly in some of the round-celled varieties, is quite abundant and arranged in a wide-meshed net, in the meshes of which the cells lie. These spaces are called alveoli, and this variety of structure has acquired importance from the general resemblance which these tumors have to the well-defined and characteristic alveolar structure which many of the carcinomata exhibit. It is true that occasionally the resemblance is very close indeed, but usually the sarcomata present a more or less intimate relation between the cells and basement substance. The cells usually do not simply lie in the cavities, but are often attached to the intercellular substance, which not seldom sends finer trabeculæ into the alveoli between the cells. Sometimes a careful shaking of sections in water is necessary to reveal the characters of the reticulum. The cells, moreover, are usually, though not always, distinctive in character. This form of tumor is, in some cases at least, determined, as above stated, by the new formation and peculiar arrangement of the blood-vessels. Tumors of this kind are not common, but

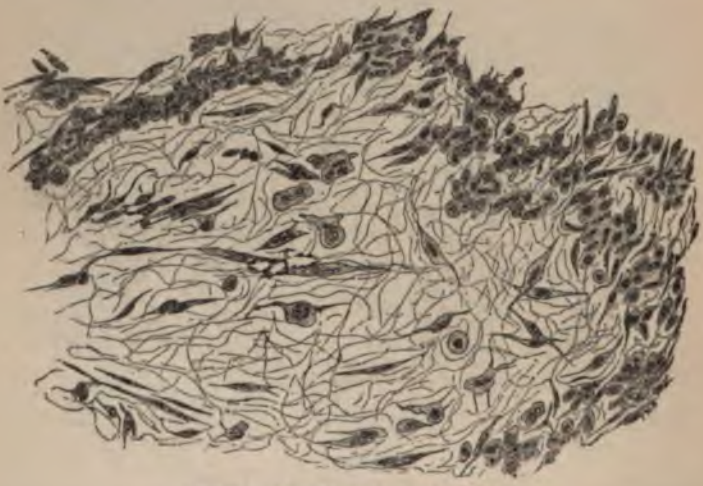

FIG. 55. MYXO-SARCOMA OF PHARYNX.

may occur in the skin, lymph nodes, bones, and pia mater. They are usually very malignant.

In addition to the above more or less well-defined forms of sarcoma, there exist various modifications which have received special names. Thus sarcomata in which cysts form, either by the softening of tissue by degeneration, or by the dilatation of gland ducts by pressure, or by the new formation of tissue in gland ducts or alveoli which dilate with the growth of the tumor, have received the name of *cysto-sarcomata.*

Mucous degeneration is frequent in the various forms of sarcoma. A combination of myxoma and sarcoma—myxo-sarcoma—is common (see Fig. 55).

Combinations of sarcoma with fat tissue, *lipo-sarcoma;* with glandular structures, *adeno-sarcoma* (Fig. 56); with cartilage, *chondro-sarcoma;* with muscle tissue, *myo-sarcoma;* and with various other tissues, are of frequent occurrence. Some forms of *psammoma,* or "brain sand," found chiefly in the dura mater, are *fibro-sarcomata* which have undergone calcification, the lime being deposited in lamellated masses of various shapes within them.

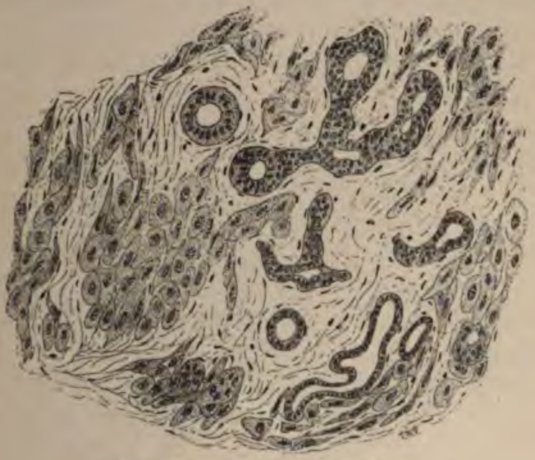

FIG. 56.—ADENO-SARCOMA OF PAROTID.

Some of the soft papillomata and warts, and occasionally the polypi of the mucous membranes, belong to the type of sarcoma or myxo-sarcoma.

The so-called *chloromata,* which have been found in a variety of places in the body, but are rare, are apparently forms of sarcoma. Chloroma is characterized by a greenish color the nature of which is not known.

Sometimes in various forms of sarcoma, as in other tumors, the endothelial cells lining the lymph spaces appear to proliferate, giving rise to a variety of rounded, elongated, or reticular structures, which somewhat resemble a typical gland formation. Such tumors are sometimes called *adenoid sarcomata,* but they require more careful and extended study before they can be definitely classified. Some of them belong to the tumors of the next class.

ENDOTHELIOMA.

Under the name of endotheliomata are grouped a number of tumors which on the one hand are closely related to the sarcomata in genesis, and in some cases in appearance, while on the other hand some of them so closely resemble some forms of carcinoma as to be difficult of distinction from them. The endotheliomata originate in that form of connective-tissue cells called endothelium, and seem to develop by a proliferation of these. Sometimes the cells of the endotheliomata resemble closely the normal endothelium; sometimes, however, they differ considerably from them, being occasionally very large, often thick and irregular in shape, and even nearly cylindrical or cuboidal. They are associated with a more or less abundant vascular stroma, which may be alveolar in formation. In this case, as in alveolar sarcoma, it may often be seen that the cells have an intimate relationship to the trabeculæ of the stroma. Sometimes the cells of the endotheliomata are packed together in dense concentric masses (Fig. 57), which may have a glistening appearance, and such tumors are sometimes called *cholesteatomata.* Although, for the most part, the peculiar glistening appearance of these tumors is due to the closely packed thin cells which compose them, they not infrequently contain crystals of cholesterin, sometimes in large quantities, which may share in producing their characteristic appearance. But the cholesterin may be absent or present in small amount.

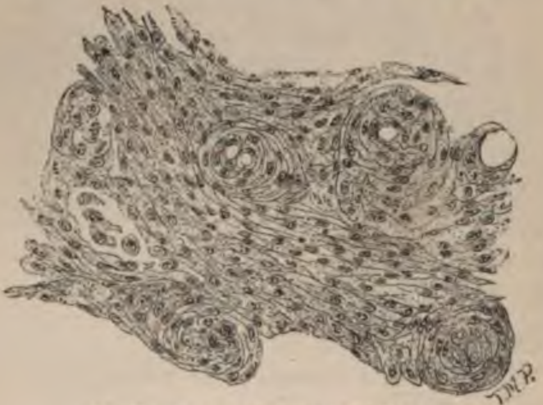

Fig. 57.—Endothelioma of Dura Mater.

The endotheliomata may occur of considerable size and be nodular, or they may be multiple, numerous small tumors being scattered over the surface of the part in which they grow; they may even form a thicker or thinner pellicle over surfaces, or cause adhesions between adjacent organs. They may form metastases. They occur in the dura mater and pia mater, in the pleura and peritoneum, and have been described in the lymph nodes, ovary, liver, brain, and testicle.[1]

LIPOMA.

Lipomata are tumors formed of fat tissue. The fat tissue occurs in lobules and is similar to normal fat, except that the cells and lobules are usually larger. There may be little connective tissue in the tumors, when they are very soft, almost fluctuating—*lipoma molle*—or there may be so much as to give the tumor considerable firmness—*fibro-lipoma.* They may be in part transformed into mucous tissue—*myxo-lipoma.* Cartilage not infrequently develops in them, or they may undergo partial calcification.

Occasionally the blood-vessels are very abundant and dilated—*lipoma telangiectoides.* They are usually sharply circumscribed, but may infiltrate surrounding tissues. They are not infrequently pediculated. They sometimes grow to enormous size, and may ulcerate.

They are usually isolated, but may be multiple. They are the most common of tumors, occurring usually in the subcutaneous or other fat tissue. They may occur in the mucous membrane of the gastro-intestinal canal, in the peritoneum, more rarely in the dura mater, kidney, liver, and lungs. They are benign tumors, not forming metastases, but they may be deleterious by ulceration or gangrene, and when not fully removed may exhibit local recurrence.

CHONDROMA.

These tumors, composed of either of the physiological forms of cartilage, are usually hard, but sometimes quite soft. The cells do not present the same uniformity in size, shape, number, and relative position that they do in normal cartilage. Sometimes they are very large, spheroidal, and grouped in masses, and again small and far apart. They are frequently fusiform or branching. Fibrillar connective tissue in varying quantity is usually present in the chondromata, either as a capsule, or running in bands between the nodules of cartilage, or passing in fascicles into them (Fig. 58). The cartilage may change to mucous tissue,[1] forming *myxo-chondroma* (Fig. 59); the cells may undergo fatty degeneration or they may calcify or ossify. Chondromata frequently form a part of mixed and complex tumors.

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[1] This class of tumors is unsatisfactory, for, as will be seen, we have no very definite morphological characteristics which cover all cases, and their relations to other tumors are so close that we often need to know the seat of the growth and something of its genesis before we can arrive even at a measurably definite determination of it, and even then we may fail. This indefinite state of affairs arises from the fact that we do not yet know enough about the normal relationships between endothelial and other connective-tissue cells; and, furthermore, we do not yet know exactly to what extent the progeny of one class of cells may grow to resemble or become identical with those of another class.

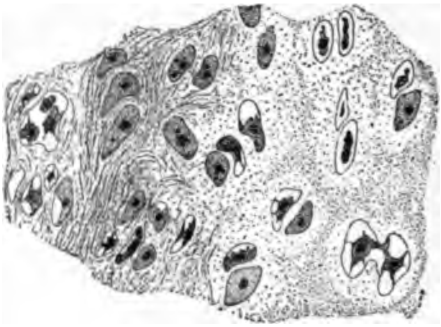

FIG. 58.—CHONDROMA OF SUBCUTANEOUS CONNECTIVE TISSUE.

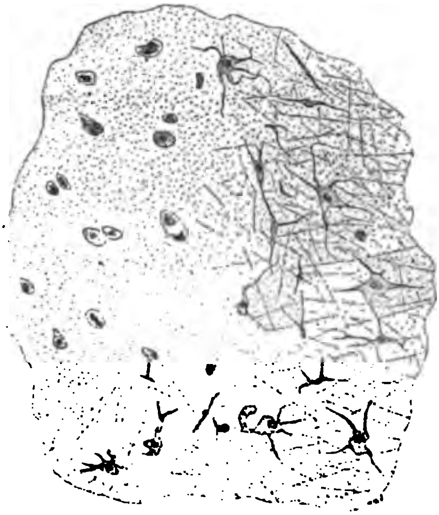

FIG. 59.—MYXO-CHONDROMA OF CERVICAL REGION.

They may form in connection with bone or cartilage, and are often traceable to irregularities in fœtal development. Or they may occur in soft parts where cartilage is not normally present, as in the parotid, testicle, mamma, and ovaries, where they are apt to be mixed with other tissues; or in subcutaneous connective tissue and fasciæ.

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[1] This change of one form of tissue into another is called *metaplasia*, and is not uncommon among the members of the connective-tissue group, as well as in the tumors which are formed after their types.

They are in general benign tumors, but metastases sometimes occur, most frequently in the lungs, sometimes in the heart.

Small hyperplastic growths on the surfaces of cartilage are called *ecchondroses.*

OSTEOMA.

The formation of bone in the body in abnormal places occurs quite frequently and under a great variety of conditions. It is on this account not easy to define the term osteoma, and it is frequently difficult to determine whether or not a given mass of new-formed bone is an osteoma or not. Bone tissue often occurs in tumors of the connective-tissue group as a secondary or complicating structure—*osteo-fibroma, osteo-chondroma, osteo-sarcoma,* etc. It may occur in muscles as a result of certain exercises, or as a result of a peculiar inflammatory process (see Lesions of the Muscles), or it may occur in connection with chronic inflammation in a variety of tissues. A circumscribed mass of abnormal bone, not of inflammatory origin, may be called an osteoma. Small masses of new-formed bone of various shape, projecting from a bony surface and frequently of inflammatory origin, are usually called *osteophytes.* Bony tumors projecting from the surface of bones are frequently called *exostoses.*

An osteoma may be loose in texture, consisting of bone tissue similar to cancellous tissue, or it may be denser, resembling compact bone tissue, or it may be very hard and dense like ivory, so-called *ivory exostoses.* The difference between these forms lies chiefly in the varying number and size of the vascular and medullary spaces which they contain.

Osteomata may develop in connection with the bone or periosteum, which is most frequently the case, or, independently of bone, in soft parts.

New-formed bone has been found in the soft parts of the body, in the brain substance, dura mater and pia mater; in the pleura, diaphragm, and pericardium; in the skin, choroid, air passages, lungs, and penis, and in other places. To what extent some of these bone formations may have been due to inflammatory action it is not possible to say, and it is quite probable that the fuller knowledge of the future may show relationships between the development of certain tumors and some forms of chronic inflammation which we do not now recognize.

The growth of the osteomata is, as a rule, slow. They are benign tumors, and are not infrequently multiple.

GLIOMA.

The gliomata are developed in connection with the characteristic connective-tissue framework of nerve tissue, the neuroglia, which in structure many, though usually not all, of its cells closely resemble. Small cells with inconspicuous bodies and numerous delicate branching processes are most characteristic; but in connection with these there is usually a greater or less number of small spheroidal cells with proportionally large nuclei (Fig. 60). It is usually necessary to shake sections in water or carefully tease fragments of the tumor in order to see the characteristic neuroglia or so-called "spider" cells (Fig. 61). These tumors may contain very numerous and frequently dilated thin-walled blood-vessels. They may be very soft or moderately hard; and especially when occurring in the substance of the brain are frequently not sharply outlined against the adjacent normal tissue. They usually occur singly, and are comparatively slow in growth.

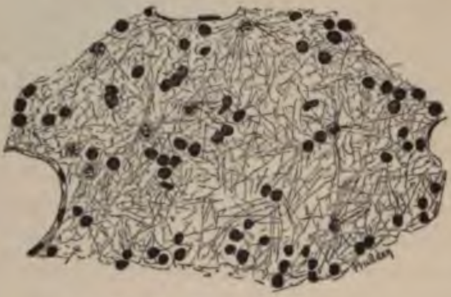

FIG. 60.—GLIOMA OF BRAIN.

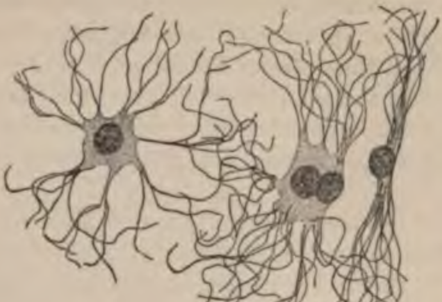

FIG. 61.—NEUROGLIA OR "SPIDER" CELLS FROM GLIOMA OF BRAIN.
Teased specimen.

They are very apt to be complicated with other tumor tissue, forming *glioma-myxoma*, *glio-sarcoma*, etc. Owing to the abundance of thin-walled blood-vessels and the softness of the growth, they are liable to interstitial hæmorrhages, and may then, when occurring in the brain, readily be mistaken for ordinary apoplectic clots. They are liable to fatty degeneration. They usually occur in the brain, spinal cord, and in the optic and other cerebral nerves. The so-called gliomata of the retina are usually small spheroidal-celled sarcomata.

Pure gliomata are benign tumors, though in their most common combination with sarcoma they may be very malignant. Their usual situation, however, is such as to make them almost always significant, although technically they are benign tumors.[1]

MYOMA.

Tumors composed of muscular tissue are of two kinds, following the two physiological types of muscle tissue, the non-striated and the striated.

I. *Leiomyoma, Myoma-levicellulare.*—The characteristic elements of these tumors are fusiform, smooth muscle fibres, with elongated or rod-shaped nuclei. These are packed closely together, frequently interlacing and running in various directions, and are intermingled with a variable quantity of more or less vascular fibrillar connective tissue (Fig. 62). When, as is not infrequently the case, the connective-tissue elements are present in large amount, the tumor is called *fibro-myoma.* It is not always easy in sections to distinguish between these tumors and certain cellular fibromata, but the characteristic shape of the isolated cells and their nuclei, together with their uniformity in size, will usually suffice. These tumors are frequently infiltrated with lime salts, and, owing to their density and lack of blood-vessels, they not infrequently degenerate, forming cysts or becoming gangrenous. They may occur singly or multiple, are usually of slow growth, may be large or small, and are benign. They may occur wherever smooth muscle tissue exists.

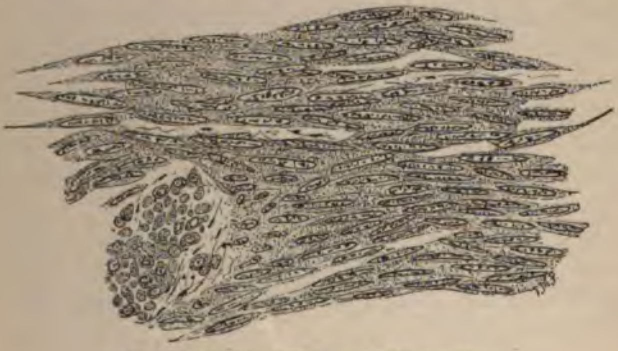

FIG. 62.—MYOMA OF UTERUS. Leiomyoma.

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[1] Our knowledge of the normal neuroglia is still too meagre to permit us to understand very thoroughly this class of tumors, and to separate it as precisely as could be wished from certain of its allies among the abnormal connective-tissue growths.

They are most frequently found in the uterus, where they are often multiple. They may occur in the wall of the gastro-intestinal canal, and have been seen in the bladder and in the skin of the nipple and scrotum. The so-called hypertrophies of the prostate, so frequent in advanced life, are usually leiomyomata of the interstitial muscle tissue of that gland.

II. *Myoma striocellulare* or *Rhabdomyoma.*—In these rare tumors, striated muscle fibres are the characteristic elements. They very rarely compose a great part of the tumor, but are intermingled with other elements, fibrillar connective tissue, spindle-shaped and spheroidal cells of various forms, which often appear to be incompletely developed muscle cells. They are not infrequently associated with sarcomatous tissue. Blood-vessels and sometimes nerves are also present. The muscle fibres differ, as a rule, from normal striated muscle fibres in their arrangement, which is usually quite irregular, and also in size, being in general smaller than normal fibres, although varying greatly. The sarcolemma is either absent or incompletely developed. These tumors are usually small or of moderate size, and are supposed to originate from inclusions of cells destined to form muscle tissue in places where they do not belong.

In the heart and certain other muscular parts, small circumscribed masses of striated muscle tissue have been described, and are sometimes called *homologous rhabdomyomata.* But genuine heterologous rhabdomyomata are, in almost all cases thus far recorded, confined to the genito-urinary organs, kidney, ovary, and testicles. The writer (T. M. P.) has described an exceptional case of rhabdomyoma occurring in the parotid gland.[1] These tumors, when not associated with other and malignant tumors, are benign, and are of much greater theoretical than practical interest.

NEUROMA.

A true neuroma is a tumor containing new-formed nerve tissue. Such tumors are comparatively rare. Tumors developed in the connective tissue of nerves and composed usually of fibrous or mucous tissue are common, and are frequently called neuromata, but they should be called fibromata or myomata, etc., of the nerves, or *false neuromata.* The true neuromata are of two kinds, *ganglionic* or *cellular neuromata* and *fibrillar neuromata,* depending upon the character of nerve tissue which they contain. The ganglionic neuromata are found associated with other structures in certain of the teratomata in the ovaries, testicles, and in the sacral region; they also occur in the gray matter of the brain.

The fibrillar neuromata are, according to Virchow, of two kinds, *myelinic* and *amyelinic,* depending upon whether the nerve fibres which they contain are medullated or not. The *neuroma myelinicum* is the more common and the best understood. The medullated nerve fibres in these tumors are associated with fibrillar connective tissue, and are usually curled and intertwined in a most intricate manner. They occur either singly or multiple on the peripheral nerves. They may occur in considerable numbers as nodular tumors on the branches of a single nerve trunk, or they may form an irregular, diffuse, nodulated enlargement of the nerve branches—*plexiform neuroma.* These neuromata may or may not be painful. They not infrequently form at the cut ends of the nerves in amputation stumps. They are benign tumors, never forming metastases.

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[1] American Journal of the Medical Sciences, April, 1883.

The false neuromata (Fig. 63) are myxomata, or fibromata, or sometimes myxo-sarcomata of the nerve sheaths or intrafascicular connective tissue, and may occur singly or multiple. In the latter case, they may affect the branches of a single nerve trunk, or they may be found on nearly all the cerebro-spinal peripheral nerves. The writer (T. M. P.) has described a case (Fig. 64) in which over eleven hundred and eighty-two distinct tumors were found distributed over nearly all the peripheral nerves of the body.[1] The nerve fibres in these tumors may be crowded apart by the new growth and considerably atrophied; or, in cases in

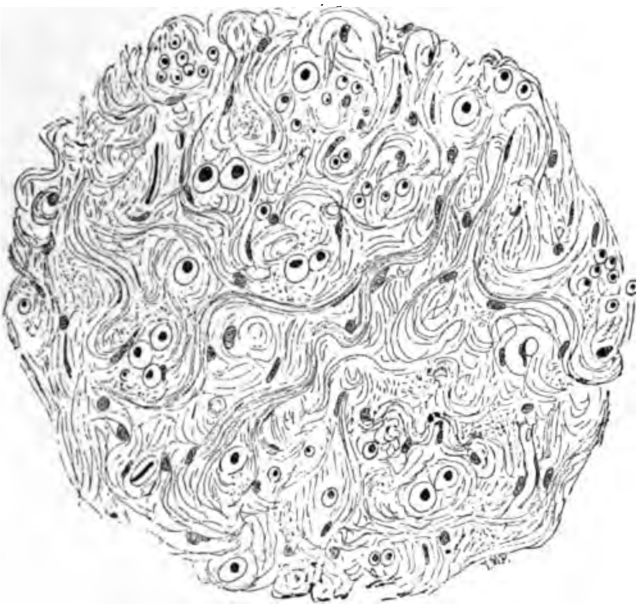

FIG. 63.—FIBROMA (FALSE NEUROMA) OF LUMBAR NERVE.

The fibrous tissue is loose in texture and in places œdematous, so that it considerably resembles mucous tissue.

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[1] American Journal of the Medical Sciences, July, 1880

which the tumor is composed of soft tissue, as in myxoma or the soft fibroma, they may pass through or around the tumor entirely unchanged. The multiple false neuromata are in many cases congenital.

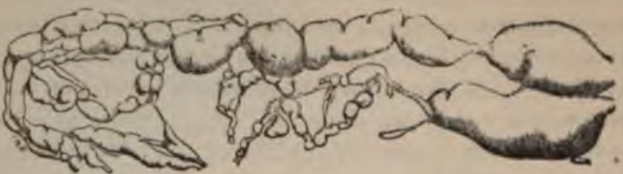

FIG. 64.—MULTIPLE FIBROMATA (FALSE NEUROMATA) OF PNEUMOGASTRIC NERVE. One-quarter natural size.

ANGIOMA.

Angiomata are tumors consisting in large part or entirely of new-formed blood-vessels or blood-cavities. In many tumors of various kinds, the new-formed or the old blood-vessels may be very abundant or prominent by reason of their dilatations; the blood-vessels of otherwise normal tissues may also be largely dilated, thus simulating vascular tumors. These are, however, not true angiomata, although sometimes reckoned among them, and in many cases closely allied to them. Such are the so-called arterial varix, or cirsoid aneurisms, and hæmorrhoids. *True angiomata* are of two kinds:

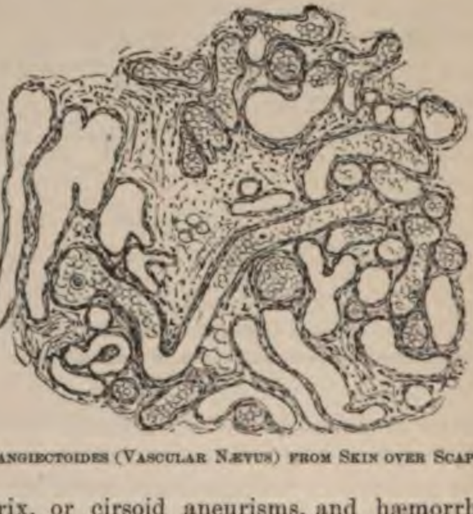

FIG. 65.—ANGIOMA TELANGIECTOIDES (VASCULAR NÆVUS) FROM SKIN OVER SCAPULA OF CHILD.

I. Those formed largely of capillary blood-vessels with either thin or thickened walls, imbedded in a more or less abundant connective-tissue stroma. These are called *simple angiomata* or *angioma telangiectoides*. The walls of the vessels in these tumors are frequently dilated or pouched, and usually form a tangle of curled and intertwined vessels. They occur most frequently in the skin or subcutaneous tissues (Fig. 65), usually about the face, and may project above the general surface or be on a level with it. Such are the so-called *vascular nævi* or *strawberry marks*, which are usually congenital. They are sometimes sharply circumscribed, and sometimes merge imperceptibly into the surrounding skin. They sometimes occur in the mucous membranes, in the mamma, bones, and brain. They are benign tumors, never forming metastases, but may be associated with sarcomata.

II. The second form of angioma, called *angioma cavernosa*, consists largely of a series of intercommunicating, irregular-shaped larger and smaller blood-spaces lined with endothelium, and surrounded by a variable quantity of fibrillar connective tissue, which may contain smooth muscle cells (Fig. 66). They resemble the erectile tissue of the corpora cavernosa of the penis and clitoris. They are apparently formed by a dilatation of old and new-formed capillaries and veins. They are sometimes erectile and sometimes pulsating, and are not infrequently multiple. They may be seated in the skin and subcutaneous tissue, forming the so-called projecting nævi, or in internal organs. They are often found in the liver and less frequently in bone, the brain, spleen, uterus, kidney, intestines, bladder, and muscles. They are usually of little significance, though they may give rise to hæmorrhages.

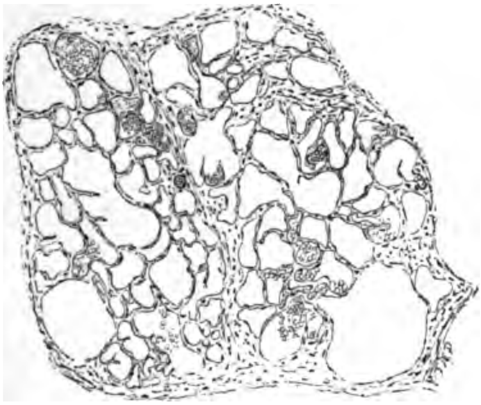

FIG. 66.—ANGIOMA CAVERNOSA OF LIVER.

LYMPHANGIOMA.

These tumors consist of dilated lymph channels, which either preserve approximately the general shape of the original lymph vessels, or are distinctly cavernous in character, or even cystic (Fig. 67). They probably originate in part in new-formed, in part in old lymph channels. A strict distinction between tumors formed by a dilatation of preformed and new-formed lymph channels is not possible, owing to the very primitive character of some of the ultimate lymph spaces and our lack of knowledge of their exact relations to adjacent parts.

In the lymphangiomata, there may be much or little connective tissue between the dilated channels, which are usually filled with a translucent or milky fluid resembling, and probably identical with, the normal lymph. These tumors are usually congenital, but are sometimes acquired. They usually occur in the skin as soft, sometimes considerably, sometimes but slightly elevated tumors, and may occur in the tongue—some forms of so-called macroglossia. They are benign tumors, but may rupture, giving rise to a serious lymphorrhœa.

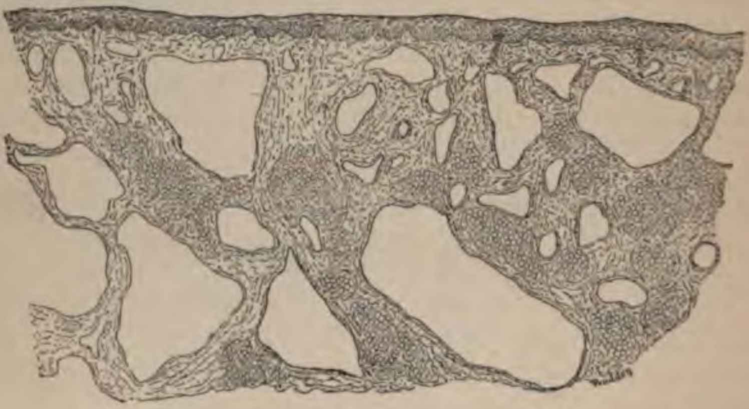

FIG. 67.—CONGENITAL LYMPHANGIOMA FROM ARM OF CHILD.

TUMORS IN WHICH EPITHELIAL CELLS ARE PREDOMINANT OR CHARACTERISTIC ELEMENTS—EPITHELIAL TUMORS.

I. *Adenoma.*
II. *Carcinoma.*

General Considerations.—The tumors thus far described in detail, with the exception of the gliomata, are formed on the type of tissues which develop from the parablast. The epithelial tumors, on the other hand, originate in one or other of the layers of the archiblast, and we have accordingly two series of criteria by which to describe and identify them: first, morphological, and, second, histogenetic criteria.

While in the main, in the normal body, the general distinctions between epithelial and other tissues are fairly well marked, there are still particular cases, especially those in which epithelial tissues are in process of physiological growth or rejuvenation, in which the distinctions are quite ill-defined. When we remember the rapid growth of many tumors, the tendency to incomplete formation of their cells, theirdiverse seats, and the various complicating conditions under which they originate and develop, it does not seem strange that the exact limitations of this class of tumors are not easy to fix, nor that they seem sometimes to merge into one another and into tumor tissues belonging to other classes. If epithelial cells, under all circumstances, had a definite and characteristic structure, or if, on the other hand, we could always know whether a given cell group originated in epithelium or not, the matter of distinguishing between tumors of this and other classes would be simple and easy enough. As it is, in some cases both morphological and histogenetic criteria fail us, and the clinical history and gross appearance are not characteristic. Such cases, which are indeed rare, but which do sometimes occur, suggest to us the possibility that the desirability of accurate classification has led us into seeking distinctions which Nature herself has not sharply drawn. While these difficulties in special cases must be acknowledged, the distinctions are in the main definite enough, and very useful both for clinical and scientific purposes.

Epithelial tumors always contain, in addition to the more or less characteristic cellular elements, a connective-tissue stroma which gives them support and carries the vessels. This stroma may be sparse or abundant, may contain few or many cells, is sometimes arranged in irregular fascicles or bands, and very frequently forms the walls of well-defined, variously shaped spaces or cavities called *alveoli,* in which the epithelial cells lie. The epithelial cells, in most cases, lie along the walls of the alveoli without an intimate connection with them, as is the case in the alveolar sarcomata. They are, moreover, packed together without more intercellular substance than the usual cementing material common to epithelial cell masses. In this lack of fibrillar intercellular substance within the alveoli, and in the loose relationship between the cells and the alveolar walls, lie in many cases the chief morphological distinctions between certain carcinomata and alveolar sarcomata.

In certain of the epithelial tumors, there is a reproduction of typical gland tissue of various kinds, depending upon the seat and conditions of growth of the tumor. Such tumors are called *adenomata.* A simple hypertrophy of a gland, or an increase in its size by excessive growth of its interstitial tissue, do not constitute an adenoma. There must be an actual new formation of more or less typical gland tissue. This is not always or frequently of exactly the same character as the gland tissue in which it originates, and always exhibits a certain lack of conformity to the type in structure and mode of growth. The alveoli and ducts usually have a lumen and sometimes a membrana propria, but the cells may differ in shape from one another and from those of the gland from which they spring.

Epithelial tumors in which there is no close conformity to a glandular type, but a lawless growth of various kinds of more or less typical epithelial cells in the meshes of an old or new-formed connective-tissue stroma, are called *carcinomata.*

It will readily be seen that there must be a border region between the adenomata and carcinomata where conformity to the glandular type merges into the lawlessness of growth characteristic of carcinomata. In this border region, a certain degree of individual bias must be permitted in assigning a name to the new growth. In some cases a sharp distinction cannot be made, or the tumor may share in the characteristics of both, and then we very properly make use of the term *adeno-carcinoma* or *carcino-adenoma.*

I. ADENOMA.

The structure of the cellular elements of these tumors and their arrangement into acini and ducts vary even more than do those of the normal glands whose types they follow. The acini usually possess a more or less well defined lumen and membrana propria (see Fig. 68). The adenomata sometimes merge into the surrounding tissue, or are continuous with the gland tissue in which they originate; sometimes they are distinct in outline and incapsulated. The interstitial tissue is sometimes abundant, sometimes sparse, and may contain few or many cells. The irregularities of their growth often lead to the stoppage of the lumina of their ducts and the formation of cysts. They may undergo mucous metamorphosis, and may become sarcomatous.

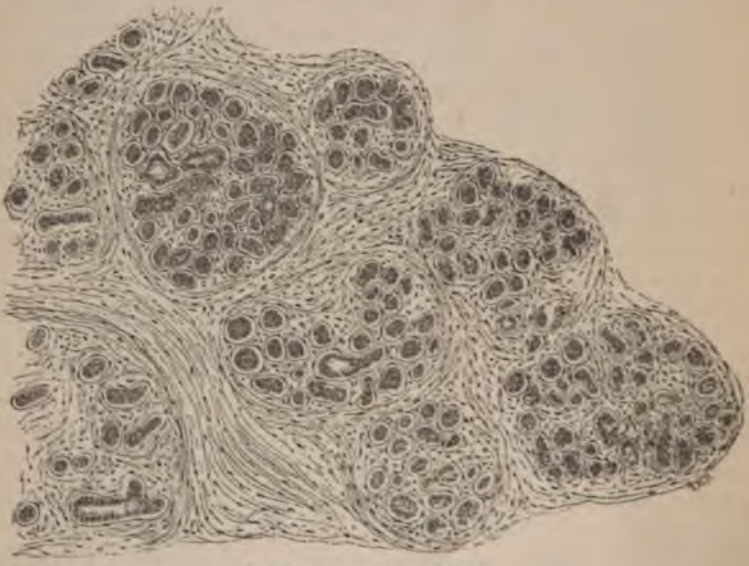

FIG. 68.—ADENOMA OF MAMMA.

Adenomata occur in the mamma, ovary, liver, kidney, thyroid, salivary, and lachrymal glands, and in the caruncle; in the mucous membrane of the nose, pharynx, stomach, intestine, and uterus; and occasionally in the sebaceous and sweat glands of the skin. The so called multilocular cystomata of the ovary are among the most important of the adenomata. There are numerous papillary and polypoid growths, in gland ducts and on mucous membranes, in which there is an actual new formation of gland epithelium, but this is usually secondary to a primary growth, beneath the epithelial layer, of some other tissue, such as fibrous or mucous tissue, and the new growth of gland epithelium simply keeps pace with the growth of the latter, to which it serves as an investment. Such growths are sometimes classed among the adenomata, but do not, strictly speaking, belong there.

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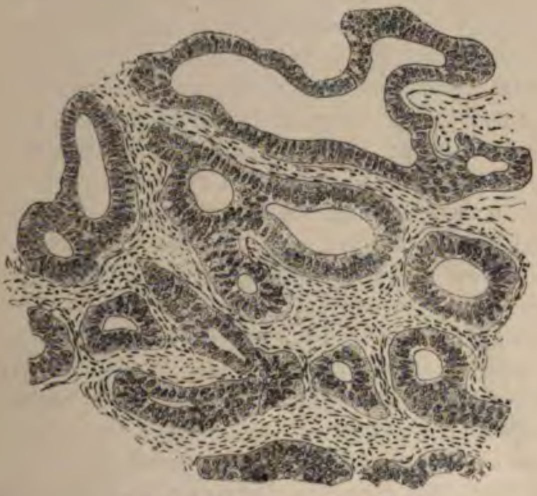

FIG. 69.—ADENOMA OF STOMACH.
A form which is on the border-line of carcinoma.

The adenomata are in general benign tumors, being slow of growth and localized, but there are very important exceptions. Some of the adenomata of the stomach and intestines belong to the most malignant of tumors in rapidity of local extension, in the formation of metastases, and the development of cachexia. Certain of the adenomata of the mamma and thyroid are also very malignant. It should be remarked, however, that, as a rule, the malignant adenomata are those which, in structure, lie close upon the border-line between tumors of this class and carcinomata (see Fig. 69), and by such observers as incline to lay more stress upon clinical than morphological distinctions they are usually classed among the latter.

CARCINOMA.

These tumors are composed, as above stated, of a connective-tissue stroma, forming more or less well-defined communicating spaces or alveoli, in which lie variously shaped epithelial cells arranged in an atypical manner. The stroma, containing few or many cells, may be, especially in the advancing portions of the tumor, composed largely of the old connective tissue of the part. It may, however, be entirely new formed. The cells which lie in the spaces or alveoli bear sometimes a very close, sometimes but a very general resemblance to epithelium.

It was formerly believed that new epithelium might be formed, both from old epithelial cells and from the connective-tissue cells, and possibly from white blood-cells, and among many observers this belief still exists and has never been disproved. Still, within the last twenty years, the opinion that new epithelial cells in tumors arise exclusively from old epithelium has found general acceptance, and for very good reasons. No one has actually seen an epithelial cell originate under the microscope, and until this can be done our beliefs must rest upon indirect observations. In the first place, all the epithelial structures in the embryo originate in the archiblastic layers, that is, in those layers which are largely characterized by the presence of epithelium (see page 121). In regeneration, after an injury in the adult, a study of the successive phases of the process shows that new epithelium is always formed in continuity with the old, and apparently by a proliferation of old epithelial cells. Epithelial tumors are almost exclusively found in parts normally containing epithelium, and frequently the new growth can be distinctly seen to be continuous with the old cells.

The occurrence of primary epithelial tumors in parts of the body in which epithelium does not normally occur, as in bone and the lymph nodes, has been recorded, but these may have been metastatic tumors, in which the primary tumor was small and overlooked, or they may have been displaced embryonic germs, which, according to Cohnheim's hypothesis (see page 119), would explain their heterologous occurrence. These possibilities of error should be taken into the account in the apparently exceptional cases, and it is to be remarked that these are becoming less and less as our knowledge increases and our technical facilities for research improve.

A considerable number of the tumors formerly described as heterologous primary carcinomata are now known to be formed by proliferation of endothelium, and hence to belong to another class, although closely resembling the carcinomata in structure.

The occurrence of primary carcinoma of the peritoneum, pleura, and pericardium, which is not infrequent, was for a long time inexplicable, because it was, and to a large extent still is, believed that the flat cells lining these great body cavities are true endothelium, and closely related in origin, as they are in structure, to the genuine endothelium of the blood and lymph vessels, etc.

But recent embryological researches have shown that this belief is not well founded. It was formerly thought that the great serous cavities were large lymph vessels formed by the splitting apart of the connective-tissue layers of the mesoblast. But we now know that the great primitive body cavity, which after a time becomes divided into the pleural, pericardial, and peritoneal sacs, is originally an outgrowth from the alimentary canal. The epithelium of the alimentary canal, however, is of archiblastic origin (see page 121), while the connective tissue, blood and lymph vessels are developed later from the parablast. Genetically, therefore, the so-called endothelial cells lining the pleural, pericardial, and peritoneal cavities are of archiblastic origin and belong among the epithelium. Thus a fuller knowledge of the histogenesis of the cells lining the great body cavities has shown us that the occurrence of primary carcinoma in these cavities is not only not in contradiction with the principle of the epithelial origin of carcinoma, but strongly confirmatory of it.

A great practical difficulty in the description, and to beginners, in the recognition of the carcinomata and their varieties, lies in the great diversity in shape which their cells present. It should be always borne in mind that the shape of cells depends in part upon their inherited tendencies in growth, which we cannot see under the microscope, but to a greater degree upon the varying conditions of nutriment and pressure to which they are exposed during life. In the normal body, these conditions conform to a certain standard, so that cells of a given kind at a given stage of development are approximately similar.

In tumors, however, the lawlessness and lack of fixed conditions in growth are such that we may have many young and atypical so-called indifferent forms of cells; while even the adult forms may depart widely from normal shapes. Thus, in cylindrical-celled carcinomata there are many fully developed cells which are never cylindrical; there are many others not fully developed which are quite indifferent in form, looking just like many other young cells—cells which are not, but which are destined to become, epithelium. Finally, we have the cells produced by ordinary inflammatory processes about and within the tumor, which acts like an irritating foreign body. Thus it is that there is no characteristic cancer cell, as was formerly supposed. Some of them are typical and some not, and the more typical ones may look just like normal epithelial cells, and the atypical ones just like simple inflammatory cells, or young connective-tissue cells, or white blood-cells. It is always in the topography, together with the general characters of the cells and the situation of the growth, that we must seek for the evidences of its nature.

The carcinomata are very prone to local extension, the advancing tumor cells in the periphery making their way through the lymph spaces and forming new foci (Fig. 70). Metastasis is of frequent occurrence in some forms, and takes place chiefly, though not exclusively, through the lymph vessels, frequently involving adjacent or remote lymph nodes. The growth of the tumor cells in the lymph vessels, either in the immediate vicinity of the original tumor or following metastasis in a distant part of the body, may cause these to become distended, and, on free surfaces like the pleura and peritoneum, to form a whitish, elevated network. Transverse sections of such distended lymph vessels are shown in Fig. 71. The secondary tumors are in the main similar in general structure to the primary foci, but may vary from them in vascularity and the abundance of the stroma, or in the shape of the cells. The carcinomata are, as a rule, malignant tumors, but the different forms vary much in this respect. They are liable to fatty, colloid, mucous, and amyloid degeneration, and are especially prone to ulceration, to hæmorrhage, and simple inflammation (Fig. 72).

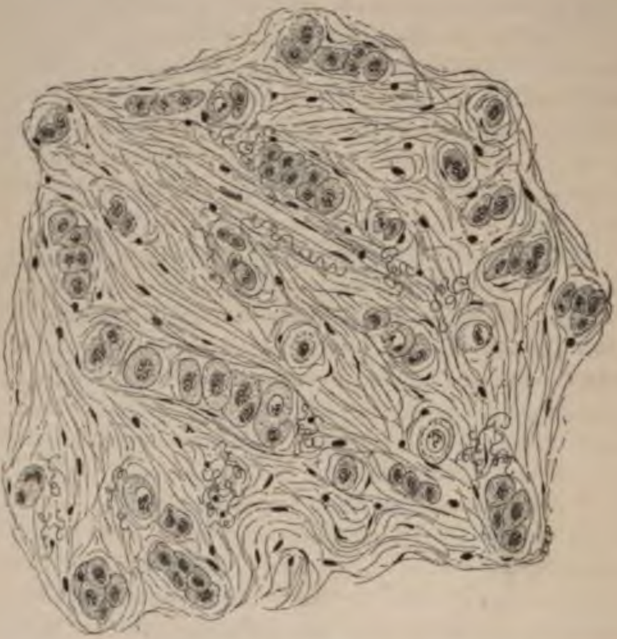

FIG. 70.—CANCER CELLS INFILTRATING THE TISSUE IN THE VICINITY OF A TUMOR. From carcinoma mammæ.

They may become partially calcified, and are not infrequently combined with other forms of tissue in the mixed tumors.

They are more frequent in middle and old age than in the young, but they may occur at any age.

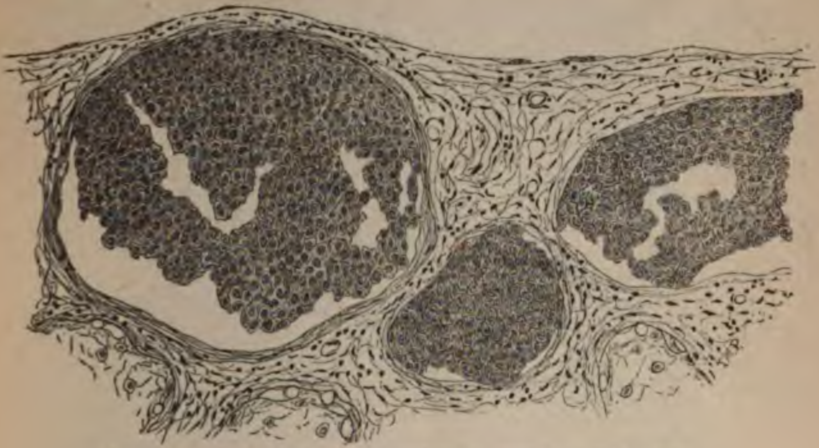

FIG. 71.—METASTATIC CARCINOMA IN LYMPH VESSELS OF THE PLEURA. The primary tumor was in the liver.

Forms of Carcinoma.—In certain cases of carcinoma which occur in the skin and in some mucous membranes, the cells present the structure and general characters of the epithelium of the part in which they occur; and since here the tendency of the cells as they approach the surface is to become flattened or squamous, these tumors are called *squamous* or *flat-celled carcinomata*, or simply *Epitheliomata*.

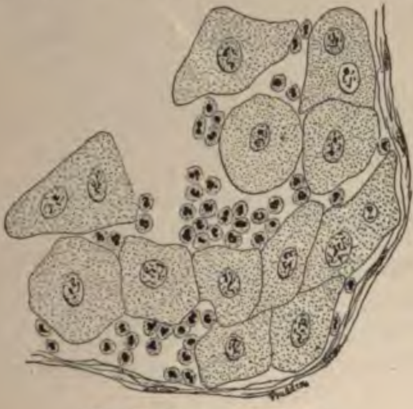

FIG. 72.—CARCINOMA UTERI. Showing accumulation of pus cells between the epithelial cells of the tumor.

In another class of tumors, such as frequently occur in the gastro-intestinal canal and uterus, the cells are more or less cylindrical in shape, forming a palisade-like lining to the irregular alveoli; such tumors are called *Cylindrical-celled Carcinomata*, although here again many of the cells are not cylindrical at all, but may have a great variety of forms.

There is a third and very common form of tumor, in which the epithelial cells have no constant characteristic shape, but vary as much as do the cell forms in the various glands of the body. Such tumors are conveniently classed together as *gland-celled carcinoma*, or *Carcinoma simplex*.

In addition to these forms, there are several others which depend for their characteristics upon various metamorphoses or degenerations, or upon the preponderance of one or other of the anatomical constituents of the growth. It will be most convenient to give a brief description of these various kinds, one after another, with the understanding that they are not absolute specific forms, but are simply varieties which it is convenient to recognize for clinical as well as anatomical purposes.

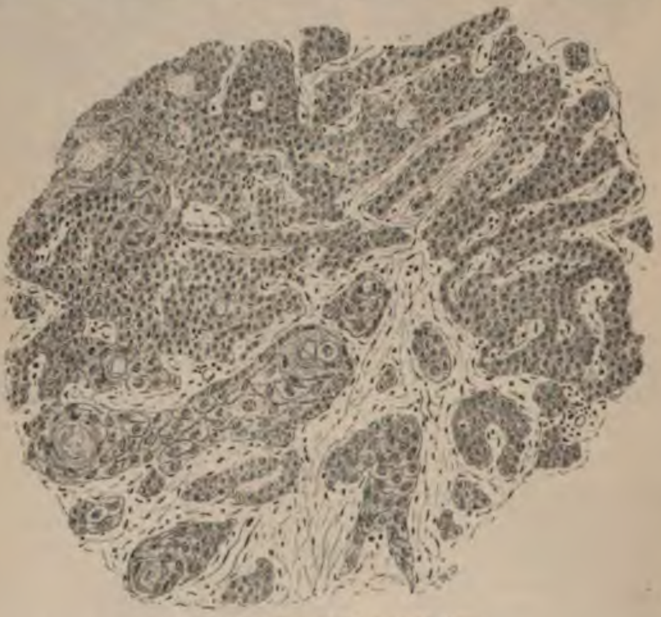

FIG. 73.—EPITHELIOMA OF THE NECK.
Shows epithelial pearls, spined cells, and reticular masses of variously shaped epithelial cells.

Flat-celled Carcinoma, or Epithelioma.—These tumors occur in the skin and in the mucous membranes, which are covered with squamous epithelium. The cells present all of the various forms which normally exist in these parts—the cuboidal and polyhedral cells of the rete Malpighii, as well as the more superficial flattened forms (Fig. 73). Frequently the spined cells, or so-called "prickle cells," are largely reproduced. Having to a certain extent the same life history as the cells in which they originate, many of the tumor cells become dry, thin, and horny, like the epidermis cells, as they grow older; and since their growth and changes often occur within the old lymph spaces of the affected tissue, or in the new-formed alveoli, the cells are sometimes

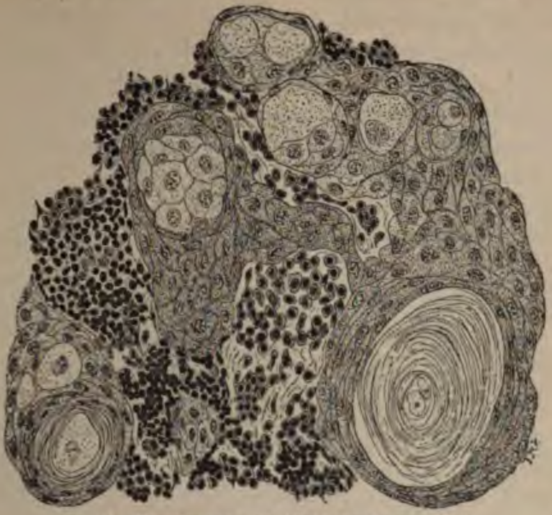

FIG. 74.—EPITHELIOMA OF AXILLARY LYMPH NODE.

This metastatic tumor was secondary to a large epithelioma of the back of the hand, Fig. 75. The small cells with darker nuclei are the cells of the lymph node.

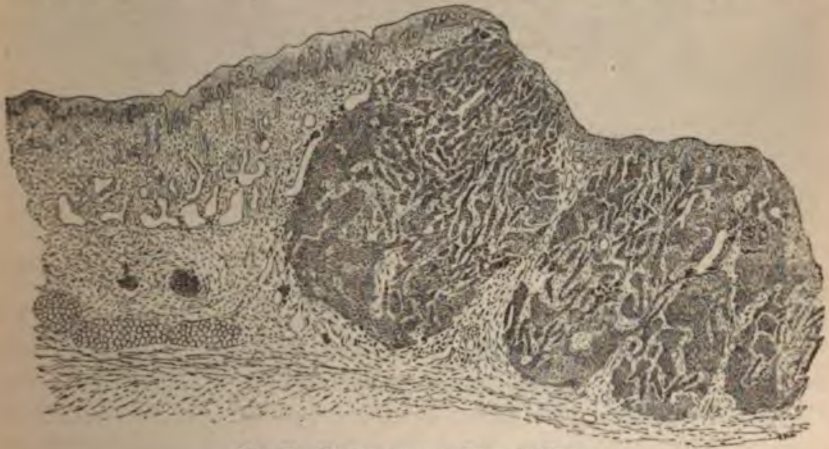

FIG. 75.—EPITHELIOMA OF BACK OF HAND.

The flat tumor occupied nearly the entire back of the hand, and was ulcerating at the centre. The figure shows the edge of the tumor and a portion of the ulcer. The papillæ of the skin over the edge of the growth are hypertrophied, and the tissue about infiltrated with small spheroidal cells.

packed together in spheroidal, concentric masses called "epithelial pearls," which may sometimes be seen with the naked eye upon or near the surface of the growth (Fig. 74). The new cell masses may be large or small, may be separated by much or little stroma; often form reticular masses, and may infiltrate the tissues deeply or remain near the surface; or may project above the surface, forming wart-like or papillary growths. These tumors frequently ulcerate on the surface, and the skin about them is apt to become thickened (see Fig. 75).

They are most apt to occur in the skin, especially in those parts in which it becomes continuous with mucous membranes—lips, external nasal openings, eyelids, labia, and glans penis—and are frequent in the mouth, œsophagus, vagina, and about the cervix uteri.

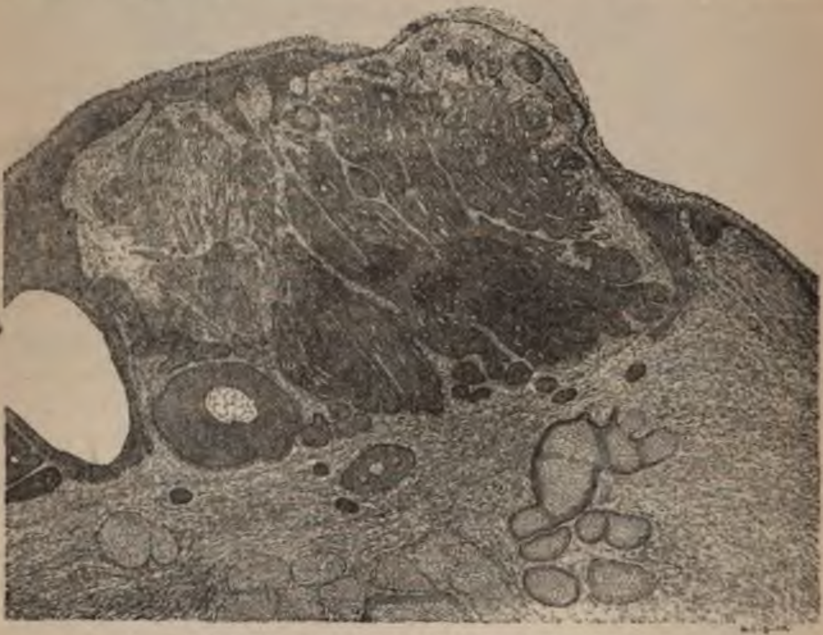

FIG. 76.—A SMALL EPITHELIOMA OF THE SIDE OF THE NOSE, × 80.

There are also carcinomata of the skin, composed of cuboidal cells arranged in tubules or masses, which do not follow the type of the epithelium of the skin, but rather that of the sweat glands or sebaceous glands. These tumors are found most frequently on the nose and eyelids (Figs. 76 and 77).

Epitheliomata are apt to recur if not thoroughly removed, and may form metastases, but in general they are the least malignant of the carcinomata. The prognosis is in most cases good if there is early and complete removal.

Cylindrical-celled Carcinoma.—These tumors, closely allied to some forms of adenoma, occur in the stomach, intestines, and uterus (see Fig. 69). The cells may be only in part cylindrical, the remainder having various shapes, and all being loosely or closely packed in larger or smaller alveoli. They may have much or little stroma. They merge imperceptibly into the next class:

Gland-celled Carcinoma, or Carcinoma simplex.—These, which are by far the most frequent of the carcinomata of internal parts, are characterized by the alveolar structure and by the absence of any special characteristic shape in the cells, which may be spheroidal, polyhedral, fusiform, or cuboidal. They may or may not resemble the epithelium of the gland in which they originate. They are usually nodular tumors, and may be hard or soft. If the stroma is abundant and dense, and preponderates over the cellular elements, the tumor is usually hard and is called *scirrhus* or *fibro carcinoma* (Fig. 78). If, on the other hand, the cellular elements largely preponderate, the tumor is usually soft, and, if it do not contain too many blood-vessels, may have a general resemblance to brain tissue, and is then called *encephaloid* or *medullary cancer;* or, better, *Carcinoma molle* (Fig. 79). These are among the most malignant of the cancers. The intercellular tissue may become so abundant as to nearly obliterate the cellular elements, but it is doubtful if carcinoma ever undergoes spontaneous cure in this way. These

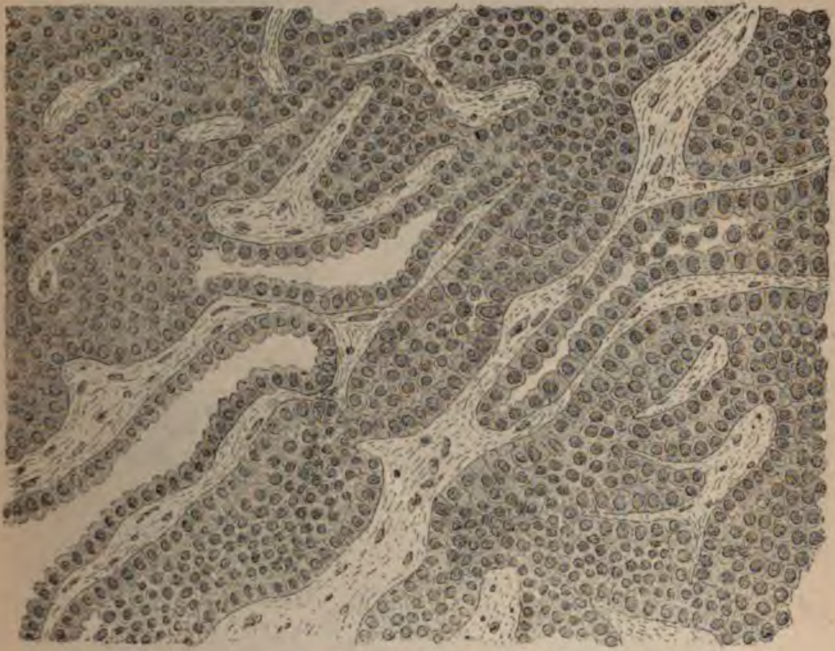

FIG. 77.—EPITHELIOMA OF NOSE, × 550.
A portion of the tumor shown in Fig. 76 more highly magnified.

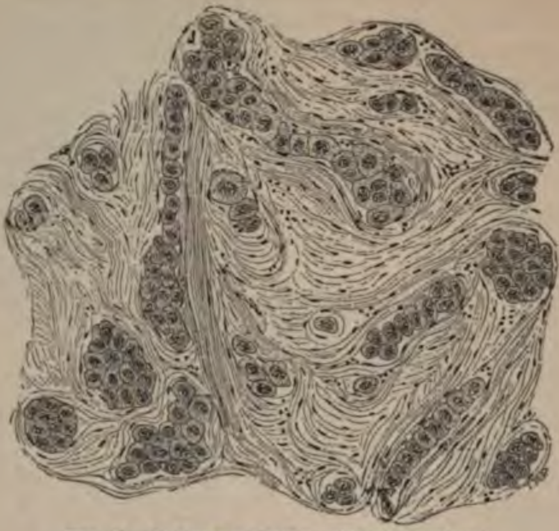

FIG. 78.—CARCINOMA MAMMÆ. (Scirrhus variety.)

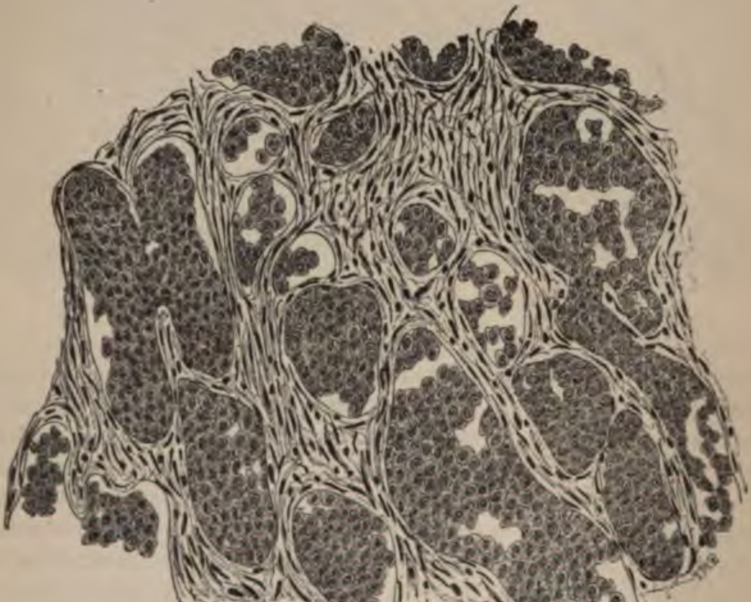

FIG. 79.—MEDULLARY CARCINOMA OF STOMACH. (Carcinoma molle.)

tumors may be hard in one portion and soft in another. They may contain very many blood-vessels, *C. telangiectoides*. They occur as primary tumors in the mamma, liver, thyroid, salivary, and prostate glands, in the pancreas, kidney, testicle, and ovary.

Colloid Carcinoma.—The cells of certain cancers, especially of the gastro-intestinal canal, may suffer a more or less complete infiltration with a translucent material somewhat resembling gelatin, and called colloid, whose nature is not well understood. Sometimes this infiltration is only partial, when the protoplasm of the cells may be more or less incroached upon by the translucent droplets of the colloid material; but in other cases over large areas the cells are partially or entirely destroyed, and replaced by the new material, so that the alveoli of the tumor are distended by it, and their walls appear very distinct in the midst of the colloid substance (Fig. 80). In such cases, the alveolar structure of the tumor is sometimes very evident to the naked eye, and these tumors are therefore often called *alveolar carcinoma*. Sometimes only a part of the tumor is affected in this way.

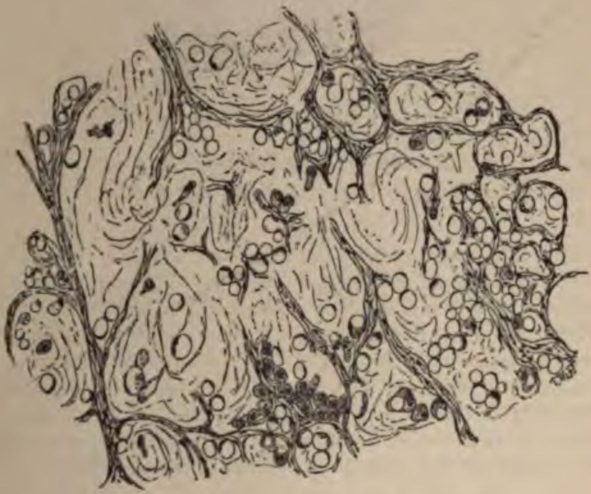

FIG. 80.—COLLOID CARCINOMA OF RECTUM.

Carcinoma myxomatodes.—The cellular elements of carcinomata may suffer mucous softening, and thus larger and smaller cysts containing a mucous fluid are sometimes formed. To this form of metamorphosed tumor the above name is sometimes applied, but it more properly belongs to cancers in which the stroma is composed of mucous tissue (Fig. 81). Such tumors are most frequently found in the gastro-intestinal canal and mamma.

Melano-Carcinoma.—Tumors of this class are rare, and are characterized by the presence of a variable quantity of black or brown pigment particles either in the stroma or the cells. They are usually soft and malignant, and most frequently occur in the skin.[1]

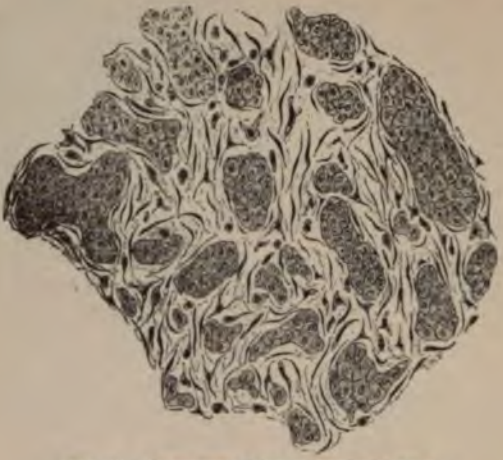

FIG. 81.—CARCINOMA MYXOMATODES MAMMÆ.

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[1] *Bibliography.*—The most extensive and important work on tumors, containing a vast store of information, is that of *Rudolph Virchow,* "Die krankhafte Geschwülste." It is not completed, and is somewhat old, but is still invaluable as a work of reference. The section on tumors in v. Pitha and Billroth's work on surgery ("Handbuch der allgemeinen u. speciellen Chirurgie"), which comprises the first section of the second volume, by *Dr. Lücke,* is very complete. A valuable bibliography and digest of recent observations on tumors will be found in the last edition of *Birch-Hirschfeld's* work on pathological anatomy ("Lehrbuch der pathologischen Anatomie"), vol. i.; also in *Ziegler's* "Lehrbuch der path. Anat.," Band i., 5th ed., 1887.

PART III.

MORBID ANATOMY OF THE ORGANS.

THE NERVOUS SYSTEM.

THE MEMBRANES OF THE BRAIN.

THE DURA MATER.

The dura mater is a dense connective-tissue membrane, which serves the double purpose of a periosteum for the inner surface of the cranial bones, and of an investing membrane for the brain. It is itself but poorly supplied with blood-vessels, but it contains the large venous sinuses which carry the blood from the brain. Lesions of the dura mater, therefore, are apt to be associated with lesions of the cranial bones, of the pia mater, or of the venous sinuses.

In young children the dura mater adheres closely to the inner surface of the cranial bones, in adults it is more readily detached, and in old persons it is again more adherent. Chronic inflammation of the external layers of the dura mater also renders it more adherent to the bones.

Hæmorrhages.—We find extravasations of blood between the dura mater and the cranial bones, in the substance of the membrane, and between the dura mater and the pia mater.

The hæmorrhages in the substance of the dura mater are usually small and of little consequence

The hæmorrhages between the dura mater and the pia mater occur with chronic pachymeningitis, or are derived from the vessels of the pia mater.

The hæmorrhages between the dura mater and the cranial bones are produced by blows and injuries of the head. They are often of considerable size. separate the membrane from the bones. and may compress the brain. They are often associated with laceration of the brain. and hæmorrhages between the dura mater and pia mater.

The pressure on the head of the infant in labor may produce, in addition to the extravasations of blood between the bones and the pericranium, additional extravasation between the bones and the dura mater.

Thrombosis of the venous sinuses is not uncommon. Any inflammation of the dura mater is liable to produce it; injuries and inflammations of the brain and pia mater, of the cranial bones, of the middle ear, and of the scalp, may also produce thrombosis. The changes in the blood produced by the exhausting and infectious diseases may produce thrombosis of the venous sinuses, as they do of the veins in other parts of the body. There are also rare cases in which such a thrombosis is developed without discoverable cause in persons previously healthy, and produces marked symptoms and death.

Some of these thrombi are firm, of white or red color, and apparently produce no secondary lesions.

Others are of firm consistence, but they produce softening with small hæmorrhages of portions of the brain. In these cases, the thrombus extends from the venous sinus into one of its veins, and the portion of brain belonging to this vein is softened and hæmorrhagic. Such a softening of the brain is often attended with inflammation of the pia mater.

In other cases, the thrombi are soft and puriform; fragments of them become detached and lodge as infectious emboli in the arteries in different parts of the body.

Inflammation of the dura mater is called *pachymeningitis,* and this may involve the external layers of the membrane, *pachymeningitis externa,* or the internal layers, *pachymeningitis interna.* It may furthermore be either acute or chronic. The tissues of the substance of the dura mater participate to a greater or less degree in these changes, but the chief lesions are upon the surfaces.

Acute *pachymeningitis externa* is usually secondary to injuries or diseases of the cranial bones; thus fractures of the skull, either depressed or not, ostitis, caries, suppurative inflammation of the internal and middle ear and mastoid cells, may produce it. The dura mater is usually congested, thickened, and softened, and may present small ecchymoses. The inflammation is usually suppurative. and pus may accumulate between the membrane and the bone, or in the substance of the membrane. The areas of inflammation are not usually extensive. It sometimes induces thrombosis of the venous sinuses, and sometimes gangrene of the dura mater occurs. The inflammation may extend to the inner surface of the dura mater, to the pia mater and brain, or it may remain localized and undergo resolution.

Acute *pachymeningitis interna* may be secondary to inflammation of the external surface, or it may occur as a complication in pyæmia, puerperal fever, chronic diffuse nephritis, in the exanthemata and erysipelas, or idiopathically. There is a general or circumscribed production of fibrin and pus, so that the internal surface of the membrane is lined with a layer of soft, yellow exudation.

Simple *chronic pachymeningitis* consists in the formation of new connective tissue in the dura mater, by which it becomes thicker, and in many cases abnormally adherent to the bones of the skull. This thickening may be general or circumscribed, and may involve the entire thickness of the membrane. Not infrequently, when the external layers are especially involved, firm adhesions to the skull occur, with ossification of the outer layers, so that shreds of the membrane containing little masses of bone (osteophytes) remain sticking to the skull when the membrane is stripped off.

There is an important form of chronic inflammation of the internal layer of the dura mater, called *pachymeningitis interna hæmorrhagica,* characterized by the formation of layers of new delicate connective tissue with numerous very thin-walled blood-vessels from which the blood is prone to escape. The membrane may at first appear as a delicate fibrinous pellicle, with small red spots scattered through it, or it may look like a simple reddish or brown staining of the inner surface of the dura mater. Microscopical examination shows this membrane to consist of numerous blood-vessels, mostly capillaries with very thin walls, which may be distended or pouched, and which have grown out from the vessels of the dura mater (Fig. 82). Between the vessels is a homogeneous or slightly differentiated basement substance, containing a variable number of spheroidal, fusiform, or branching cells. Red blood-cells in variable quantity, and blood pigment in various forms, frequently inclosed in the

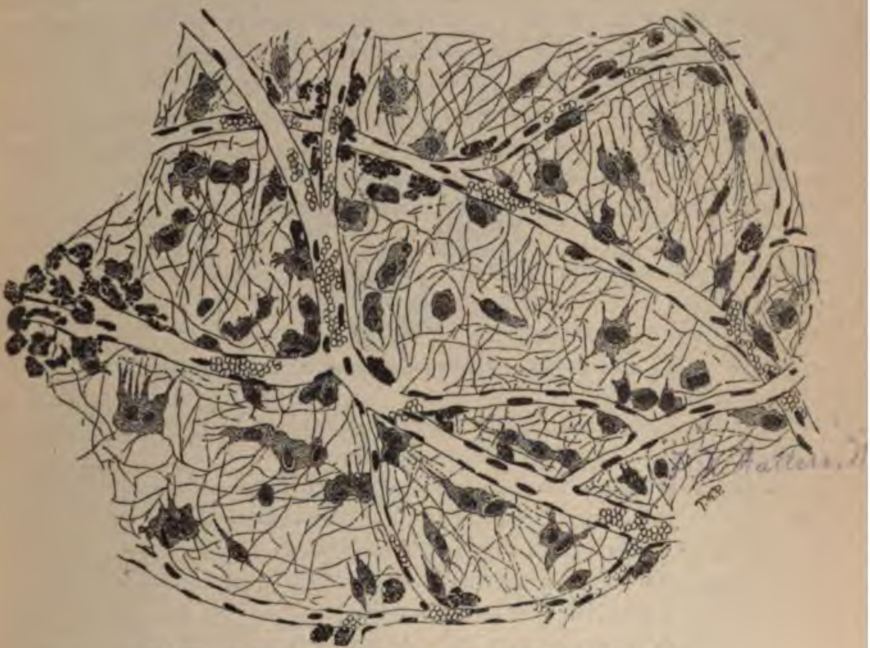

FIG. 82.—CHRONIC PACHYMENINGITIS INTERNA HÆMORRHAGICA.

new cells, and small calcareous concretions (brain sand) (Fig. 83), also lie in the intervascular spaces. In more advanced stages, the new membrane may become greatly thickened, its outermost layers being changed into dense fibrous tissue with obliteration of the vessels; while the more recently formed layers are similar in structure to those at first developed. Considerable blood usually escapes from the vessels of the new membrane by diapedesis, in all stages of its formation, and the vessels are also very liable to rupture, giving rise to extensive hæmorrhages either into the substance of the membrane or between it and the pia mater. Sometimes masses of new tissue and blood, from half an inch to an inch or more in thickness, are in this way formed, greatly compressing the brain. These new membranes are most frequently formed over the convexity of the brain, but may extend over nearly the entire surface of the dura mater. Sometimes, when old, the entire membrane, densely pigmented and firm, lies loosely beneath the dura mater without compressing the brain or giving any clinical indication of its presence. The membrane may induce chronic changes in the pia mater, with or without accompanying changes in the cortical portion of the brain.

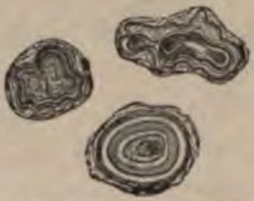

FIG. 83.—BRAIN SAND FROM PACHYMENINGITIS INTERNA.

Rarely, serum accumulates between the layers of the new membrane, and in this way cysts of large size may be formed. In rare cases, diffuse suppuration of the entire new membrane occurs.

The slighter degrees of this form of inflammation may occasion no symptoms during life. They are not infrequently found in persons suffering from various chronic brain lesions and from chronic alcoholism, but they may occur unassociated with complicating lesions. The more advanced forms of the lesion are frequently found in idiots, epileptics, etc.

Tubercular pachymeningitis may occur secondarily to that form of inflammation in the pia mater or the bones, or as a part of general miliary tuberculosis. The tubercles may be situated on either surface of the membrane or in its substance, and may be single or aggregated, forming large masses.

Syphilitic pachymeningitis manifests itself by the formation of so-called gummy tumors either upon the external or internal surface of the dura mater. These tumors may be single or multiple, and vary greatly in size. They may be accompanied by simple inflammatory changes in the dura mater in their vicinity. They may undergo suppuration with the formation of abscess; the inflammation may extend to the pia mater, inducing simple or syphilitic meningitis and adhesions between the dura mater and pia mater. The gummata may, on the other hand, when occurring on the outer surface of the membrane, cause absorption and perforation of the bones of the skull.

Tumors.—The most common tumors of the dura mater are *sarcomata*, and of these the spindle-celled forms are of more, the round and polyhedral celled of less frequent occurrence. They may grow from either surface of the membrane. Some of the round and polyhedral celled forms are soft and very vascular, and are apt to involve the neighboring pia mater and brain tissue, or the bones of the skull, which they may perforate. They sometimes project through the opening in the skull in fungous, bleeding masses.

Psammomata are small, globular tumors, often multiple and pediculated, growing from the inner surface of the dura mater. They are usually composed of tissue sarcomatous in character, and contain variously shaped calcareous concretions similar in appearance to the so-called brain sand.

Endotheliomata.—These tumors may grow inward or outward, causing pressure on the brain or absorption and perforation of the bones; they often attain considerable size. Some of these tumors somewhat resemble certain forms of epitheliomata (see Fig. 57), and have often been described as primary carcinomata.

Fibromata and *Lipomata* occur rarely in the dura mater and are of small size.

Small *Chondromata* are sometimes found connected with the dura mater at the base of the brain.

Osteomata.—In addition to the formation of osteophytes in chronic external pachymeningitis, plates, and, more rarely, globular masses of bone may be formed in the dura mater, unconnected with the bones of the skull. They are most frequently found in the falx cerebri, but may occur elsewhere. The new bone may be dense or loose in texture, and usually produces no symptoms.

THE PIA MATER.

The external surface of the brain is invested by a connective-tissue membrane which covers the convolutions, dips down into the sulci, and extends into the ventricles. This membrane is abundantly supplied with blood-vessels, and from it numerous vessels extend into the brain, so that any disturbance in the circulation of the blood in the pia mater involves a disturbance in the circulation of the blood in the brain also.

The connective tissue which makes up the pia mater is arranged in a series of membranes and fibres reinforced by elastic tissue, so arranged as to form a spongy membrane containing numerous cavities more or less filled with fluid. These cavities are continuous with the perivascular spaces which surround the vessels that pass from the pia mater into the brain.

The outer layers of the pia mater are the most compact, and are covered on their outer surface by a continuous layer of endothelial cells. This external layer of the pia mater is often described as a separate membrane called the "arachnoid," but it is really only part of the pia.

The deeper layers of the pia contain the blood-vessels. The membranes and fibres which compose the pia mater are partly coated with cells which have irregular and delicate cell bodies and large, distinct nuclei.

In all inflammations of the pia mater, the inflammatory products regularly collect in the spaces within it.

Along the borders of the longitudinal fissure, and, more rarely, on the under surface of the brain, are a number of small, white, firm, irregular bodies—the Pacchionian bodies. They vary in their size, their number, and in the extent of the surface of the hemispheres which they cover. They may perforate the dura mater, or, more rarely, the wall of the longitudinal sinus, and may produce erosions of the skull bones. They are composed of fibrous tissue and may undergo fatty or calcareous degeneration. As they are so commonly found and are not known to be of any pathological significance, they may almost be regarded as normal structures; at any rate, we do not know what causes them or their variations in size and number.

The pia mater is frequently thickened, opaque, and white, either in diffuse patches or, more commonly, along the course of the vessels. In other cases, single or multiple small white spots, of the size of a pin's head or smaller, may be seen in the membrane, not appreciably elevated above the surface, but due to localized thickening. These slight opacities of the pia mater are commonly believed to be dependent upon repeated congestions of the membrane or upon chronic meningitis, but there is no evidence that this is always the case. They are most frequently found in old persons, but may exist at any age, and do not necessarily indicate the pre-existence of disease, although similar appearances are common in the chronic insane and in drunkards.

The amount of blood contained in the vessels of the pia mater after death varies greatly, and is by no means a reliable indication of the amount present during life. In general anæmia, the vessels of the pia mater may contain little blood, but, on the other hand, they sometimes seem to contain a relatively larger amount than other parts of the body.

In œdema of the brain and pia mater, the vessels of the latter may contain but a small amount of blood.

The pia mater may be *hyperæmic* in early stages of meningitis, after death from delirium tremens, or following epileptic convulsions, from various infectious diseases, certain poisons, the presence of tumors or exudations pressing on the veins, as well as from general and local diseases of the circulatory apparatus. But whether they are overfilled or comparatively empty after death seems to depend upon the position in which the body has lain, upon the time which has elapsed between death and the examination, upon the rapidity with which the blood coagulates, and upon conditions entirely unknown to us.

Œdema.—The amount of serum beneath the pia mater and infiltrating its tissue is very variable in amount. It may accumulate as a result of atrophy of the brain substance or of venous hyperæmia, and sometimes is, and sometimes is not, accompanied by œdema of the brain substance. It may be diffuse or localized. It is not infrequent to find in hospital patients suffering from chronic nephritis, cardiac or pulmonary disease, or chronic alcoholism, a very considerable amount of serum in this situation, and yet the patient has been free from cerebral symptoms. In other cases, again, this same serous effusion affords the only explanation of grave cerebral symptoms. It is necessary to be very careful in judging of the importance of this accumulation of fluid.

It should always be borne in mind that an accumulation of fluid beneath and in the meshes of the pia mater may occur as a result of postmortem changes.

Hæmorrhage.—This may occur either into the space between the dura mater and pia mater—*intermeningeal hæmorrhage*—or in the meshes of the pia or between the latter and the brain. It may be due to injury, to rupture of aneurisms or otherwise diseased blood-vessels, to thromboses of the venous sinuses, or to causes which we are unable to ascertain. Hæmorrhages, without known cause, not infrequently occur in the substance of the pia mater in young children, but in adults they are apt to be the result of injury. Multiple ecchymoses, however, in the substance of the pia mater sometimes occur in infectious diseases and also in acute inflammation of the pia mater. Hæmorrhages in the brain substance may lead to the accumulation of blood beneath or in the meshes of the pia mater. Intermeningeal hæmorrhage in infants as a result of injury during birth is not uncommon. Small, and sometimes considerable, extravasations of blood may occur from diapedesis, and sometimes, as a result of chronic congestion, degenerated blood pigment collects along the walls of the vessels. The extravasated blood in meningeal hæmorrhage, if small in quantity, may be largely absorbed, leaving a greater or smaller accumulation of pigment at the seat of the hæmorrhage, and such pigmentations may last for a long time.

Inflammation of the pia mater is called lepto-meningitis, or simply meningitis. We distinguish acute, chronic, tubercular, and syphilitic meningitis.

Acute Meningitis occurs most frequently as the characteristic lesion of epidemic cerebro-spinal meningitis; it is a not very infrequent complication of pneumonia, Bright's disease, typhus and typhoid fever, and the exanthemata; it is secondary to injuries and inflammation of the cranial bones, of the dura mater, and of the middle ear, and it is sometimes an idiopathic lesion.

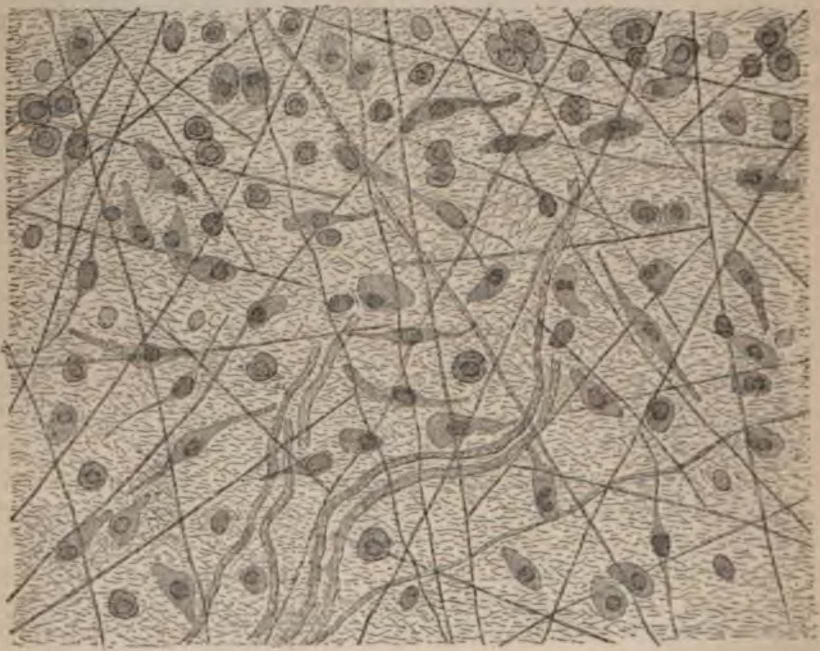

FIG. 84.—CELLULAR MENINGITIS, × 850 and reduced.

In any case of acute meningitis, the inflammation is apt to extend downward and involve the pia mater of the cord. It may also involve the ependyma of the ventricles, and cause the distention of these cavities with serum. This latter condition belongs especially to young children.

There are two anatomical varieties of acute meningitis, which give, however, the same clinical symptoms.

(1) *Acute cellular meningitis.*—The pia mater is somewhat congested, its surface is dry and lustreless, and it is somewhat opaque. These changes in the gross appearance of the membrane are not marked, and are easily overlooked, but the minute changes are more decided.

There is an abundant production of cells somewhat resembling the cells which coat the surfaces of the membranes and fibres which make up the pia mater (Fig. 84). This cell growth is general, involving the pia mater over most of the surface of the brain. The inflammation, then, is one which results in the production, not of fibrin, serum, or pus, but of new connective-tissue cells. This form of meningitis is of frequent occurrence, and is attended with the ordinary clinical symptoms of acute meningitis.

(2) *Simple acute meningitis* of the exudative form is characterized by the accumulation, chiefly in the meshes of the pia mater and along the walls of the blood-vessels, of variable quantities of serum, fibrin, and pus. Sometimes one, sometimes another of these exudations preponderates, giving rise to serous, fibrinous, or purulent forms of the inflammation. The absolute quantities, too, of the exudations vary greatly.

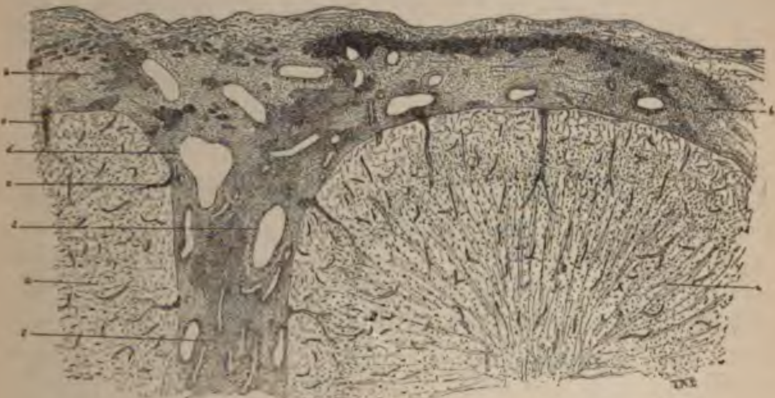

FIG. 85.—ACUTE PURULENT MENINGITIS.

a, convolutions of cerebrum ; *b*, pia mater thickly infiltrated with pus ; *c*, blood-vessels entering brain from pia and surrounded by a zone of pus cells ; *d*, congested blood-vessels of pia mater ; *e*, smaller blood-vessels of pia, around which pus cells are collected in dense masses.

In some cases, death may be caused with so slight a formation of exudation that, to the naked eye, the pia mater may look quite normal or perhaps only moderately hyperæmic or œdematous; the microscope, however, in these cases, will reveal pus cells in small numbers, and sometimes flakes of fibrin in the meshes and along the walls of the vessels. In other cases, turbid serum in the meshes of the membrane is all that can be seen, and the microscope shows the turbidity to be due to pus cells or a small amount of fibrin. Again, either with or without marked œdema of the pia mater, yellowish stripes are seen along the sides of the veins, sometimes appearing like faint turbid streaks, and at others dense, opaque, thick, and wide, and almost concealing the vessels. These are due to the accumulation of pus cells and fibrin in large quantities along the vessel, and they are best seen and most abundant around the larger veins which run along over the sulci. In still other cases, the infiltration with pus and fibrin is so dense, and thick, and general that the brain tissue, convolutions, and most of the vessels of the pia mater themselves are concealed by it. This is usually of a greenish-yellow color, and is sometimes so thick as to form a sort of cast of the brain surface at the seat of the lesion (Fig. 85). Sometimes extravasated red blood-cells are mingled with the other exudations as the result of diapedesis. Microscopical examination shows numerous white blood-cells sticking in the walls of the veins and capillaries, or the vessels may be blocked with them. It is evident that a large part of the pus cells accumulate as the result of emigration. The connective-tissue cells of the pia mater may be detached from their places or degenerated. In some cases, there are considerable accumulations of pus between the pia mater and the brain substance and along the vessels which enter the latter. More rarely pus is found upon the free surface of the membrane. The brain substance may be compressed by the accumulated exudation, so that the convolutions are flattened. The cortical portion of the brain may be simply infiltrated with serum—œdematous—or it may undergo degenerative changes, or it may be the seat of punctate hæmorrhages. Not infrequently the inflammation extends to the ventricles, which may contain purulent serum, and to the pia mater of the

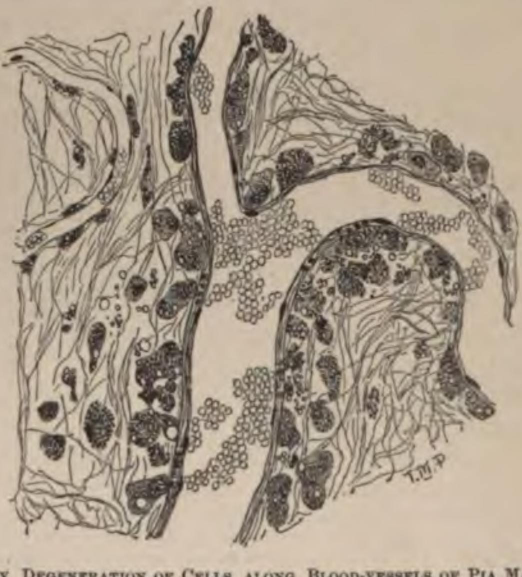

FIG. 86.—FATTY DEGENERATION OF CELLS ALONG BLOOD-VESSELS OF PIA MATER AFTER EXUDATIVE MENINGITIS.
From a child five years old.

cord. This form of inflammation is most frequent on the convexity of the brain, but may extend, or even be confined to the base. It may be localized, but frequently extends widely over the surfaces of the hemispheres. Bacteria are often present in the exudation. For their relationship to the lesions, see Cerebro-spinal Meningitis.

When recovery from acute exudative meningitis occurs, there may be fatty degeneration of the cells which have accumulated in the pia mater, particularly along the vessels (Fig. 86), and this may produce white patches in the membrane and threads along the blood-vessels, which resemble the appearance of an accumulation of exudation in the acute stage. Fatty degeneration of the blood-vessels and cells of the pia mater may also occur without acute inflammatory changes.

Sometimes, in children and young adults, the inflammatory changes in the ventricles persist for days and weeks after the subsidence of the inflammation of the pia mater.

Chronic Meningitis.—Either the pia mater at the base of the brain alone may be inflamed (basilar meningitis), or the pia mater over the convexity alone, or the entire pia mater, or circumscribed patches of the membrane. The pia mater is thickened and opaque, the thickening being sometimes very considerable. There is a formation of new connective tissue and a production of pus, fibrin, and serum; the relative quantity of these inflammatory products varies in different cases. Firm and sometimes extensive adhesions may be formed between the dura mater and the pia mater. Not infrequently the cortical portions of the brain participate in the morbid process, and we find infiltration of small spheroidal cells around the blood-vessels, thickening of the walls of the vessels, and degenerative changes and atrophy of the nerve tissue. New connective tissue may also form in the brain substance, which may become closely adherent to the pia mater. The ventricles of the brain also may contain an increased amount of serum, and may be dilated, and the ependyma may be thickened and roughened. This form of inflammation may be the result of injury or disease of the cranial bones, or secondary to chronic pachymeningitis or to inflammation of the brain substance. It may occur in the vicinity of tumors of the brain or meninges. It may be a complication of chronic diffuse nephritis or the result of chronic alcoholic poisoning. It may occur in marked form in the general paralysis of the insane.

Tubercular Meningitis.—This is especially characterized by the formation in the pia mater of miliary tubercles, associated with more or less well-marked exudative inflammation. It may occur in adults and in children, but is more common in the latter. The dura mater may be unchanged, or its inner surface may be sprinkled with miliary tubercles. The pia mater may or may not be congested; it may look dry on the surface or it may be œdematous. Usually the brain seems to fill the cerebral cavity to an unusual degree, and the convolutions are flattened. If the pia mater be œdematous, the serum may be clear or turbid with pus and fibrin. The membrane may present any of the general appearances of exudative meningitis. But always in addition to these, and sometimes without them, miliary tubercles, either widely scattered or in great numbers, may be seen, usually more abundant over the sulci than elsewhere. They are usually more abundant at the base of the brain than on the convexity, and are frequently confined to the base. Some of the tubercles are so small as to be scarcely visible or entirely invisible to the naked eye; others are as large as a pin's head or larger. They are usually most abundant along the blood-vessels, but may occur elsewhere. They may be formed in the membranous prolongations of the pia mater which dip into the sulci, around the vessels which enter the brain substance, in the choroid plexus and ependyma of the ventricles, and may exist in the spinal cord.

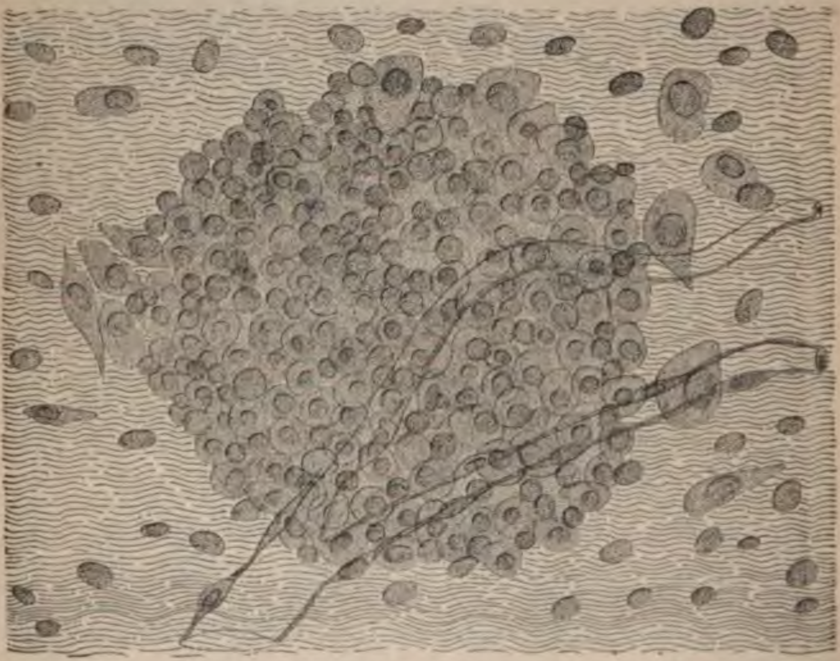

FIG. 87.—A MILIARY TUBERCLE OF THE PIA MATER, × 850 and reduced. Composed of a simple aggregation of cells.

The miliary tubercles do not all have the same structure. Some of them are simply small aggregations of round cells within the perivascular sheaths of the smaller arteries. Others are composed of small masses of polyhedral and round cells without any basement substance between them, and without any special relation to the blood-vessels (Fig. 87).

Many others have the ordinary structure of tubercle tissue, basement substance, polyhedral cells, and giant cells. These tubercles are usually situated around or near a blood-vessel, and this blood-vessel is apt to be at the same time the seat of an obliterating endarteritis (Fig. 88). This form of tubercle is also prone to cheesy degeneration (Fig. 89).

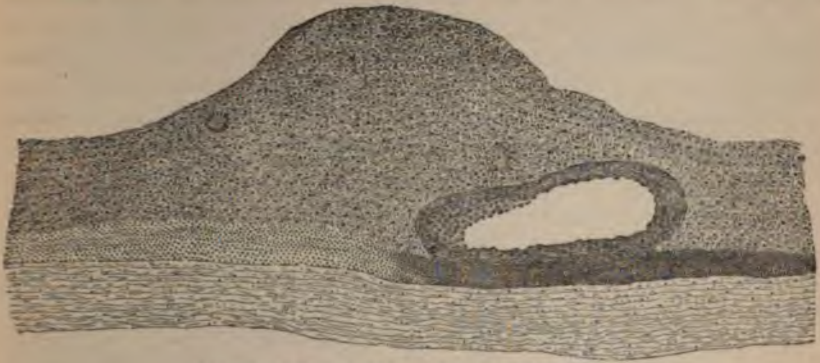

FIG. 88.—A MILIARY TUBERCLE OF THE PIA MATER.
Situated on the wall of a small artery which is the seat of endarteritis.

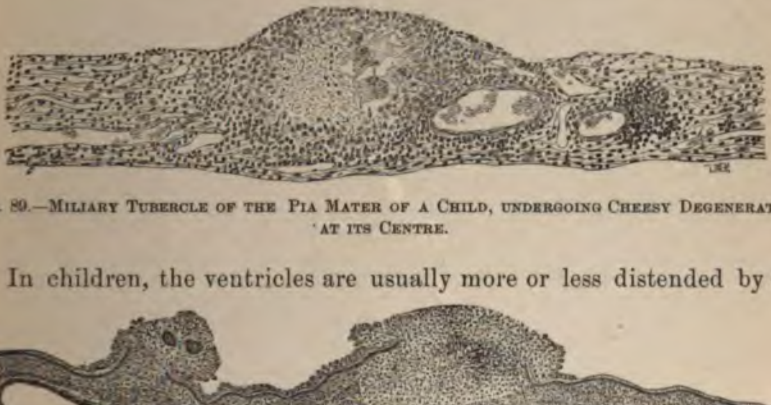

FIG. 89.—MILIARY TUBERCLE OF THE PIA MATER OF A CHILD, UNDERGOING CHEESY DEGENERATION AT ITS CENTRE.

In children, the ventricles are usually more or less distended by an accumulation of transparent or turbid serum, and the walls of the ventricles may be studded with miliary tubercles (see Figs. 90 and 91). In

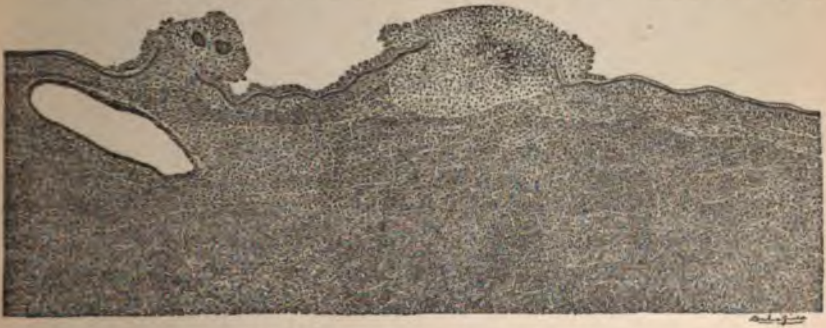

FIG. 90.—MILIARY TUBERCLES OF THE EPENDYMA OF THE LATERAL VENTRICLE, × 70 and reduced.

adults, the ventricles are less frequently involved. The brain tissue around the ventricles is often softened. The central canal of the spinal cord may also be dilated. It is the dilatation of the ventricles which causes the flattening of the convolutions, and the flattening is usually in direct proportion to the amount of accumulated fluid. Miliary tubercles in the choroid of the eye are present in a considerable proportion of cases.

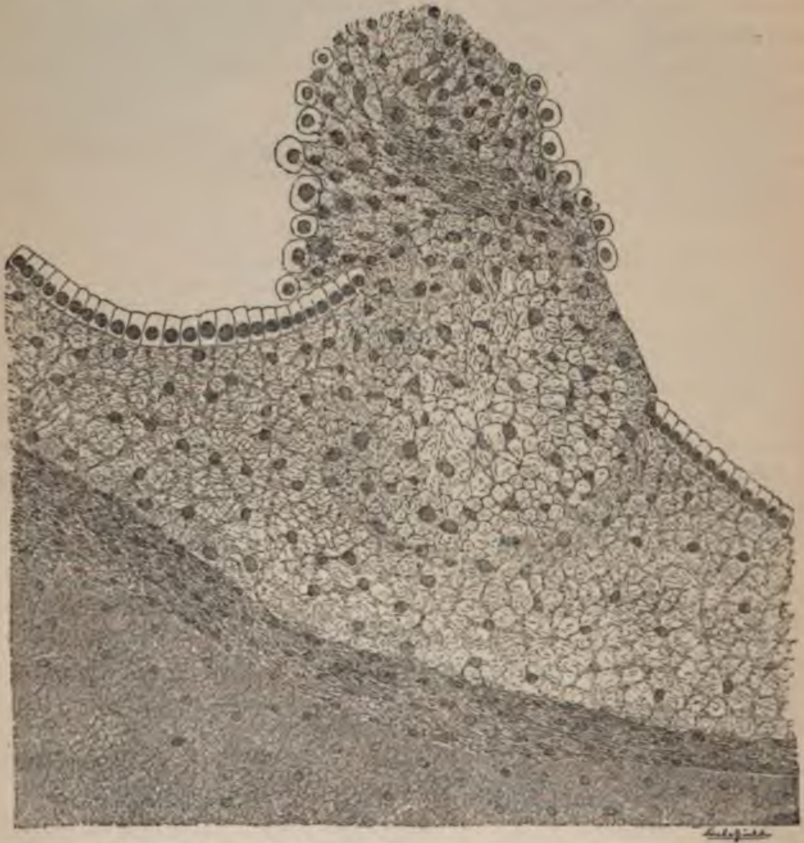

FIG. 91.—MILIARY TUBERCLE OF THE EPENDYMA OF THE LATERAL VENTRICLE, × 500 and reduced.

The cortex of the brain may be hyperæmic, and punctate hæmorrhages may be present in the cortex and in the pia mater.

In almost all cases of tubercular meningitis, there is tubercular inflammation in other parts of the body.

In adults, as in children, while the tubercular inflammation is always present, the accompanying simple inflammation may be very slight or extensive, and the degree to which it develops does not seem to depend upon the abundance of the miliary tubercles. Owing to the frequency of the dilatation of the ventricles with serum in children, the disease is often called *acute hydrocephalus.*

In both children and adults, the tubercular inflammation may produce large masses of tubercular tissue, which undergo cheesy degeneration, in the pia mater and the brain tissue.

Syphilitic Meningitis.—In this form of inflammation, which is usually circumscribed, there is a development of gummy tumors of variable size, frequently associated with simple inflammation of the membrane, either with the formation of serum, fibrin, and pus, or with the development of new connective tissue and the consequent thickening of the membrane. The gummata may form in the pia mater covering the convexity, or at the base of the brain. They may grow outward, involving the dura mater; or inward, incroaching upon or involving the brain tissue. Although usually circumscribed, the syphilitic inflammation may occur as a diffuse thickening of the membrane. The syphilitic nodules, including the gummata and new-formed connective tissue, are often very small, but may be as large as a hen's egg.

Tumors.—Aside from the Pacchionian bodies, which are sometimes so large as to simulate tumors, the most common forms of primary tumors are *sarcomata* and *endotheliomata* (cholesteatomata). Small *fibromata, lipomata, angiomata,* and *myxomata* occur, but are rare. *Lymphangiomatous cysts* sometimes occur in the pia mater, and various forms of *carcinoma* may occur as secondary tumors. The pia mater is frequently involved in tumors growing from the dura mater or the brain substance.

Variously shaped *pigment cells* not infrequently occur in the pia mater, either scattered or sometimes in considerable masses; they seem to have little pathological significance. Not infrequently thin plates of new-formed *bone* are found in the pia mater, associated with a thickening of the membrane.

Parasites.—*Cysticercus* has been observed in the pia mater.

THE VENTRICLES OF THE BRAIN.

THE EPENDYMA AND CHOROID PLEXUS.

As the lymph spaces of the pia mater and the ventricles of the brain are in communication, it might be supposed that they would share alike in the accumulation of fluids. This, however, is not the case. The membranes of the brain may be highly œdematous while the ventricles contain about the normal quantity of fluid; or, on the other hand, the ventricles may be widely dilated and the pia mater unusually dry. Many of these varying conditions may be understood by remembering that the skull and spinal canal form a closed cavity, and that accumulations of fluid in one part must be at the expense of some material occupying other parts, either blood, serum, or brain tissue. It is not always easy to see, however, exactly how the compensation occurs.

There may be an unusual amount of fluid in the ventricles of the brain as a result of post-mortem change; in connection with senile or other atrophy of the brain; or in the general vascular changes which lead to œdema of the brain; in connection with inflammation of the meninges or of the ependyma; or under conditions which we do not understand, as in some cases of congenital and acquired hydrocephalus. Accumulations of fluid in the ventricles are often called *internal hydrocephalus*, to distinguish them from accumulations in the meninges—*external hydrocephalus*.

Acute Inflammation of the Ependyma (*Acute Ependymitis*).—In this condition, which may occur by itself, but is usually associated with inflammation of other parts of the brain, the ependyma is congested, the vessels are more prominent than usual and are often tortuous. The ependyma and the adjacent brain tissue may be thickened and infiltrated with pus cells, and the surface of the ependyma covered with fibrin and pus in variable quantity. The cavities of the ventricles may contain purulent serum. Small hæmorrhages may also be present in the tissue of the ependyma. This, as well as other forms of inflammation, is more common in the lateral ventricles than in the others, but not infrequently involves the fourth ventricle. The choroid plexus may participate in the inflammatory changes of the ependyma. Tubercular inflammation of the ependyma is, as above mentioned, a not infrequent accompaniment of tubercular meningitis.

Chronic Inflammation of the Ependyma (*Chronic Ependymitis*).—This lesion, which is much more common than simple acute inflammation of the ependyma, occurs under a variety of conditions, and its nature and causation are in general very obscure. The ependyma is thicker, whiter, and more opaque than normal, so that the vessels may be nearly or quite invisible. The thickening may occur in patches or diffusely, and the surface of the ependyma may be smooth, or roughened and granular. On microscopical examination, the surface of the ependyma may be covered with the usual epithelium, but the new connective tissue which forms beneath it often raises it up in places, causing the roughness of the surface. The new tissue is usually rather loose in texture and may contain many small spheroidal cells; but it may be dense in texture and contain few cells. The brain tissue beneath the thickened ependyma may be softened or infiltrated with cells. The sides of the ventricles may be grown together in places by the adhesion of the thickened and roughened ependyma. The ventricles usually contain more serum than normal, and sometimes this accumulation is so great as to cause an enormous dilatation of them. While these are in general the prominent lesions in chronic inflammation of the ependyma, the cases vary greatly in the degree to which these changes are developed.

The accumulation of fluid and the dilatation of the ventricles being the most marked feature in all this class of lesions, they are often called *chronic hydrocephalus*, and indeed in many cases we have no evidence that the change in the ependyma is an important or even an actual primary factor.

We may, for convenience of study, consider three classes of cases of chronic hydrocephalus: first, congenital hydrocephalus in young children; second, secondary hydrocephalus in children and adults; third, primary hydrocephalus in adults.

1. *Congenital Hydrocephalus.*—The lesion may be in an advanced stage at the time of birth, or it may be scarcely evident or but moderately developed. It may progress rapidly and cause the early death of the child, or it may develop gradually or come to a stand-still. In the more marked forms of the disease, the ventricles are widely dilated and filled with serum, which is usually transparent. Not only the lateral ventricles, but also the third and fifth, may be involved; the fourth is less apt to participate in the lesion, although it is sometimes dilated, as well as the central canal of the cord.

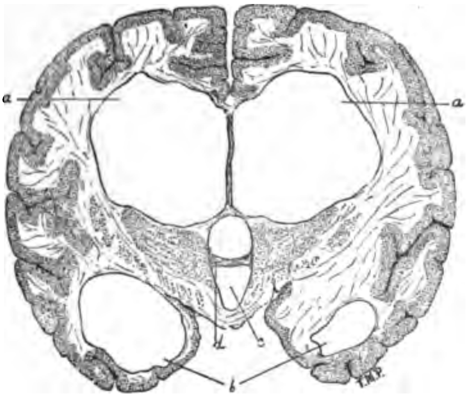

FIG. 92.—CONGENITAL HYDROCEPHALUS IN CHILD. About half natural size.

a, *a*, dilated lateral ventricles; *b*, cornua, unequally dilated; *c*, third ventricle; *d*, middle commissure.

The distention, especially of the lateral ventricles, may be so great that the brain tissue over the vertex is crowded up into a thin layer beneath the dura mater, or it may be entirely destroyed. When the dilatation of the ventricles is considerable, the convolutions are flattened (see Fig. 92) and may be almost entirely obliterated. The skull bones may be thin and bulging over the forehead and vertex; the fontanelles and sutures widely open. The ependyma in these cases is usually thick and rough, but it may be softened, and the blood-vessels may be dilated. The basal portions of the brain may be flattened, but are usually much less affected than the upper portions. The brain tissue is usually soft and anæmic.

2. *Secondary Hydrocephalus.*—This may occur in children and adults, and may be a result of epidemic cerebro-spinal meningitis, or of acute meningitis, or of chronic meningitis. It sometimes occurs in chronic alcoholic poisoning and in general paralysis of the insane. The amount of dilatation of the ventricles varies greatly in these cases, but it is never so great as in congenital hydrocephalus, and is not accompanied by the changes in the shape of the skull which form so prominent a feature in the latter disease, since the bones are firmer and the sutures united. In this form of chronic hydrocephalus, the changes in the ependyma above described are usually more or less well marked, and they may be associated with the production of fibrin and pus.

3. *Primary Hydrocephalus in Adults.*—The conditions leading to this form of lesion are not understood. It is apt to occur in persons over thirty years of age. Sometimes one, sometimes both lateral ventricles are dilated. The dilatation is usually moderate, sometimes very slight, and never as great as in congenital hydrocephalus. The ventricles usually contain transparent serum, and the ependyma is thickened and roughened. In some cases, it will be the only lesion found to account for the death of the patient.

Tumors.—The new formation of connective tissue in the ependyma, although usually diffuse, may be circumscribed and form small, projecting connective-tissue nodules, which may be reckoned among the *fibromata.* Small fibromata are sometimes detached from the walls of the ventricles and lie free in the cavity. Small *lipomata*, *angiomata*, and also *sarcomata* and *gliomata* occur rarely. *Chondromata* and *angiomata* may occur in the choroid plexus, and the latter are sometimes as large as a hen's egg. The choroid plexus is not infrequently the seat of transparent *cysts*, usually of small size; they may contain a clear fluid or colloid material, or droplets of fat or calcareous particles. A small *dermoid cyst* containing hairs has been described. These cysts have no special pathological significance.

Primary *carcinomata* are sometimes found in the ventricles.

The calcareous bodies called *brain sand*[1] occur frequently in the choroid plexus (see Fig. 83), and *corpora amylacea* may occur here and beneath the ependyma.

Cysticercus and ecchinococcus cysts are sometimes found free in the fluid of the ventricles.

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[1] The little, hard masses called *brain sand* consist of aggregations of small particles of carbonate and phosphate of lime, with a very small amount of phosphate of ammonia and magnesia. With these there is more or less organic matter.

PINEAL GLAND AND PITUITARY BODY.

Pineal Gland.—The lesions of this structure are not very common or important. It may participate in inflammatory changes involving neighboring structures. It may become hypertrophied and dense, and has thus attained the size of a walnut. Small cysts may form in it, and of very frequent occurrence is a considerable increase in the amount of brain sand, which is usually found there in moderate quantity under normal conditions.

The Pituitary Body.—This body may share in inflammatory changes occurring in its vicinity, or may be the seat of tubercular and syphilitic inflammation. The most important of the tumors which may originate in this structure are *adenomata*, usually cystic in character; these may be as large as a hen's egg. *Carcinomata* and *sarcomata* may occur, forming tumors of considerable size. Small *lipomata* and a *teratoma* have been described.

THE BRAIN.

THROMBOSIS AND EMBOLISM.

In studying the occurrence and effects of thrombosis and embolism in the brain, certain peculiarities of the circulation should be borne in mind. The arteries of the brain are in part terminal arteries (see page 57), in part such as have anastomoses among their branches. Thus the arteries which are distributed to the cortical regions form abundant anastomoses in the pia mater and are very small when they enter the brain, while those which are distributed to the basal region, and which supply the basal ganglia, are larger and do not, beyond the circle of Willis, form anastomoses with one another. Thus it is that occlusions of the arteries supplying the basal ganglia are of much more serious import, aside from the importance of the parts involved, than those passing to the cortex.

Thrombi may form in the arteries as a result of any degenerative or inflammatory process in the wall of the vessel leading to a roughening or death of its intima, or from pressure upon the vessel from without, or they may occur in vessels in whose walls we can detect no primary lesion. The most common causes are atheroma or simple endarteritis. Thrombi may also form around an embolus which does not entirely occlude the vessel.

Emboli of the cerebral arteries most commonly arise from acute or chronic endocarditis or cardiac thrombi; they may arise from aneurisms or atheroma of the aorta, from the carotid or vertebral arteries, or from the pulmonary veins. The materials constituting emboli vary greatly, depending on their mode of origin (see page 56). The effects on the brain tissue of emboli and thrombi of the arteries are essentially the same in their main features. In some cases, however, in which large emboli, usually from endocarditis, suddenly block up a large vessel, the individual may die almost instantly without other apparent lesion than the stoppage of the vessel.

In general, the first effect of the occlusion of an artery is to deprive the region to which it is distributed of blood. In arteries whose branches anastomose, as in the cortex of the brain, the affected area is soon supplied with blood by the establishment of a collateral circulation. In terminal arteries, on the other hand, the blocking of the vessel is followed, as a rule, by degenerative changes and softening in the brain tissue. The appearances which these degenerated areas present vary greatly, depending upon the stage of the degeneration and the amount of blood which may be extravasated. Dense infiltrations of the brain tissue with blood, as in hæmorrhagic infarctions from emboli in other parts of the body, do not usually occur, although considerable blood may be extravasated. Areas of softening in which there is little extravasation of blood are usually white or yellow in color (*white or yellow softening*). When much blood is present, the process is frequently called *red softening*. The tissue in the affected area gradually softens and may become diffluent.

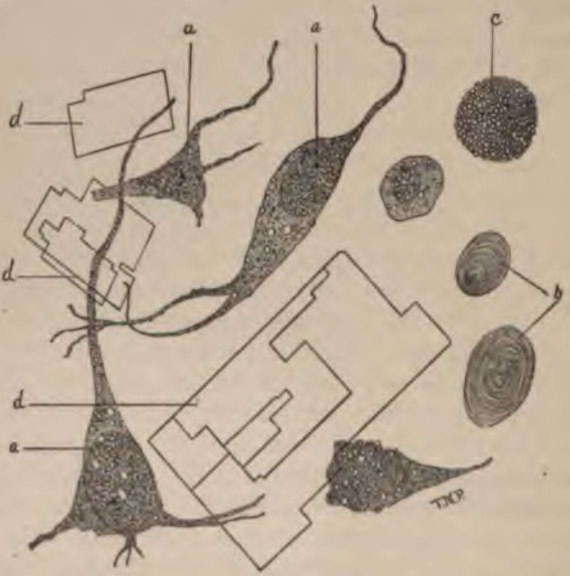

FIG. 93.—DEGENERATED CELLS, CHOLESTEARIN CRYSTALS, AND CORPORA AMYLACEA FROM BRAIN TISSUE IN EMBOLIC SOFTENING.

a, fatty ganglion cells; *b*, corpora amylacea; *c*, cell containing very large number of fat-droplets (compound granular or Gluge's corpuscles); *d*, cholestearin crystals.

Microscopically, the softened tissue is seen to consist of more or less fluid with broken-down brain tissue, fragments of nerve fibres, droplets of myelin, nerve cells, shreds of neuroglia tissue and blood-vessels, and red and white blood-cells. Then evidences of degeneration are seen in the presence of fat granules and droplets, larger and smaller cells densely crowded with droplets of fat (so-called *Gluge's corpuscles* or *compound granular corpuscles*) (see Fig. 93). Various kinds of cells and cell fragments, more or less granular and fatty, and also corpora amylacea, blood-pigment, fat-crystals, and cholestearin crystals, may be found. The walls of the blood-vessels may also be in a condition of fatty degeneration (Fig. 94). The color of the softened mass will of course depend upon the relative amounts of these elements.

The tissue may remain for a long time in the soft condition, or it may be absorbed and replaced by a connective-tissue cicatrix, which may be more or less pigmented; or a wall of connective tissue may form about it, converting it into a well-defined cyst, with or without pigmented walls; or the mass may dry and form a dense, structureless nodule. Acute inflammatory changes may occur about the dead tissue. In cases of infectious emboli, numerous abscesses may be formed in addition to their mechanical action.

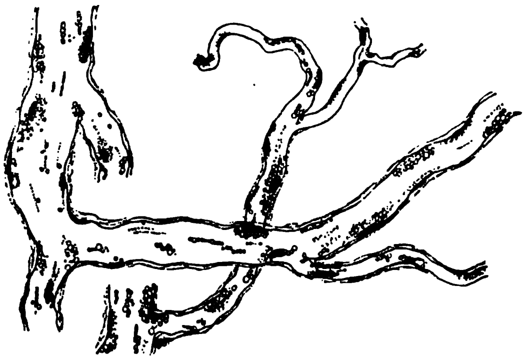

FIG. 94.—BLOOD-VESSELS FROM AN AREA OF EMBOLIC SOFTENING OF BRAIN.
The walls of the vessels, particularly the endothelial cells, contain fat-granules and fat-droplets.

Thrombi are most frequent in the internal carotids, less so in the middle cerebral, basilar, and vertebrals. They may occur, but still less frequently, in other cerebral arteries. Emboli are most common in the middle cerebral artery, next in the internal carotid, and then in the basilar. The relative frequency with which embolism occurs in the middle cerebral artery is attributable to the directness with which the blood passes into this artery from the heart. The great significance attaching to embolism of the middle cerebral artery is evident when we remember that its branches are terminal arteries, and are distributed to such important structures as the lenticular and caudate nucleus, the internal capsule, and the optic thalamus.

Hyperæmia and Anæmia.—The appearance of the brain tissue after death does not always furnish reliable indications of its blood-contents during life, though they are perhaps more to be depended on than the appearance of the meninges.

Some of the more common conditions determining *hyperæmia* which are mentioned above as influencing the meninges, apply also to the substance of the brain. In sections of hyperæmic brains, the small blood-points from the cut ends of small vessels are more numerous and conspicuous than under normal conditions, and the brain tissue, particularly the gray matter, may have a diffuse red color. If excessive, the convolutions may be somewhat flattened, and the brain tissue and pia mater may be œdematous, and the ventricles contain fluid. The congestion of the vessels may be general or localized.

Anæmia of the brain may be either local or general. It may depend upon a general anæmia or upon general disturbances of the circulation, such as mitral stenosis or regurgitation, or upon local interference with the arterial blood-supply, such as complete or partial obstruction of the arteries from thrombi, emboli, inflammatory changes, spasmodic contractions, etc., or from tumors, exudations, and blood-extravasations pressing upon the vessels from without. In œdema of the meninges, and in the presence of internal hydrocephalus, the brain tissue is apt to be anæmic. The brain tissue in anæmia looks whiter than usual, the contrast between the gray and white matter is less marked, and the small blood-points usually seen on section from divided vessels may be very inconspicuous or almost entirely absent.

Œdema of the brain tissue may accompany either hyperæmia or anæmia, and seems in most cases, though not always, to be dependent upon conditions which induce these alterations in the blood-contents of the brain. In some cases of marked impoverishment of the blood, a so-called *hydræmic œdema* of the brain is found. Very marked œdema of the brain may exist without any accompanying brain symptoms. On the other hand, persons may die comatose; this is seen with especial frequency in acute and chronic alcohol poisoning, but may occur under other conditions, and the post-mortem examination reveals nothing but œdema of the brain tissue, either with or without œdema of the pia mater.

Hæmorrhage.—Hæmorrhages in the substance of the brain may be very small and punctate, and are then usually called *capillary hæmorrhages*, or they may result in the collection in the brain tissue of masses of blood of considerable size, which are called *apoplectic foci* or clots.

These forms of hæmorrhage may be associated, or a number of capillary hæmorrhages may join to form an extensive clot.

Capillary hæmorrhages may look, on section of the brain, like the severed ends of hyperæmic blood-vessels, or the tissue about them may be more or less tinged with blood. Microscopically, the perivascular spaces will be found distended with blood, which may have escaped into them and more or less broken down the brain tissue. They may occur singly, but are frequently multiple, so that the brain tissue is besprinkled with blood-points. Degeneration of the extravasated blood may give rise in later stages to reddish or brown or yellowish circumscribed discoloration of the brain tissue, due to granules and crystals of blood-pigment intermingled with broken-down brain tissue, with more or less fatty degeneration of its elements. Capillary hæmorrhages may be due to fatty degeneration of the vessels leading to rupture; or the extravasation may be due to diapedesis, or it may depend upon conditions which we do not understand. They frequently occur in the vicinity of apoplectic clots and tumors; they may be due to thrombosis of the veins, or of the sinuses of the dura mater; they not infrequently occur in acute encephalitis, in congestive hyperæmia, in acute mania, and in delirium tremens; and they may be associated with general diseases, such as scurvy, purpura hæmorrhagica, typhus fever, pyæmia, ulcerative endocarditis, etc.; they may be associated with embolic softening.

Apoplectic foci may result from the coalescence of numerous capillary hæmorrhages: from injury, or from rupture of diseased arteries, either with or without changes in the blood-pressure. Hæmorrhages from injury to the skull may occur as well without as with fracture, and may be situated over the vertex as well as at the base of the brain, and vary in extent and seat, depending upon the character and point of the injury and the size of the vessels involved. The so-called spontaneous hæmorrhages, other than those of capillary origin, which give rise to masses of blood and broken-down brain tissue, may vary in size from that of a pea to those occupying a large part of a hemisphere. They are due in a very considerable proportion of cases to the rupture of small arterial aneurisms, but may arise from weakening of the walls of the arteries, from arteritis, atheroma, or fatty degeneration. These latter forms of disease doubtless give rise in most cases to the formation of the aneurisms whose rupture is in so many cases the immediate cause of the hæmorrhage. Aneurisms of the cerebral arteries may be as large as a pea or hazelnut, but those most frequently met with and causing apoplexy are usually small—called miliary aneurisms—and may be microscopic in size, varying from this up to that of a large pin's head or larger. They may be sacculate or fusiform, and frequently exist in considerable numbers. They may occur in any of the small arteries of the brain, but are said to be most frequent on the branches of the middle cerebral trated with pus, and outside of the walls is a zone of œdematous and softened brain tissue. If the abscess is near the pia mater, it may set up a meningitis; if it is near the lateral ventricles, it may rupture into them; if it is near the sinuses of the dura mater, it may cause thrombosis.

The incapsulated abscess has a capsule of connective tissue, and contains thin or cheesy pus.

Abscesses of the brain are usually single; they may attain a considerable size. They are most frequent in the cerebral and cerebellar hemispheres, rare in the central ganglia, the pons, and the medulla oblongata.

The most common cause of this disease seems to be chronic suppurative otitis (42.5 per cent, Gowers), while acute otitis is a comparatively rare cause. With the otitis there may also be caries of the temporal bone, suppuration of the mastoid cells, and inflammation of the dura mater. The abscess is usually situated deep in the brain; only rarely is it continuous with the inflamed dura mater and bone. When the abscess is deeply situated, and the bone and dura mater are not diseased, it is difficult to tell how the infection travels from the ear to the brain. Abscesses due to this cause are situated in the temporo-sphenoidal, the frontal, the occipital, and the parietal lobes, or in the cerebellum.

Another frequent cause of abscess of the brain is traumatism—blows or falls on the head (twenty-four per cent, Gowers). Such injuries may not hurt the skull, or may produce fractures or necrosis. There is often a considerable interval between the time when the injury is inflicted and that when the symptoms of the abscess are developed.

When the cranial bones are uninjured, the abscess is situated deep in the brain; when there is necrosis of the bones, the abscess may be superficial; when the bones are fractured, the abscess may be either superficial or deep. The abscess is regularly situated beneath the point of injury, rarely in the opposite side of the brain.

Chronic disease of the nose, either the mucous membrane or the bones, has been the cause of a few abscesses in the frontal lobes. Disease of the orbit has also given rise to abscesses in the same position. In a few cases, the abscesses have been due to caries of various portions of the cranial bones.

In a considerable number of cases (one-sixth, Gowers), no cause for the abscess has been discovered.

Very frequently in acute meningitis there is an infiltration of pus cells along the walls of the vessels which enter the brain from the pia mater; and under a variety of conditions which we do not understand, as in some cases of typhoid fever, delirium tremens, erysipelas, and under many other conditions, there are numerous and sometimes very large numbers of leucocytes scattered through the substance of the brain, sometimes around the ganglion cells, sometimes along the vessels in the perivascular sheaths.

Chronic Interstitial Encephalitis—Sclerosis.—This lesion of the brain tissue may occur diffusely, occupying an entire lobe or more or less of the whole brain, or in circumscribed small areas. It consists essentially in an increase of the connective-tissue elements, the neuroglia, and an atrophy of the nerve elements, particularly the ganglion cells and the medullary sheaths of the nerves. With these changes are usually associated the formation of Gluge's corpuscles, corpora amylacea, granular and fatty degeneration of the nerve elements, and thickening and proliferation of cells of the walls of the blood-vessels. The areas of sclerosis may be very dense and hard, or gelatinous in consistency.

The diffuse form of sclerosis is most frequently seen in general paresis of the insane, and not infrequently in the brains of drunkards.

The circumscribed form of sclerosis, *multiple sclerosis* (*sclérose en plaque*), is much more common than the diffuse form, and may occur in the brain alone, or may be associated with a similar lesion in the spinal cord. It is almost entirely confined to the medullary substance, and the areas of sclerosis vary in size from that of a pea to that of an almond. They may be few or numerous. they may be white, grayish, or grayish red in color, and are usually, but not always, sharply outlined against the unaltered brain tissue. Although in many cases the increase in the connective-tissue elements seems to be the primary lesion, and the degeneration of the nerve elements secondary to this, it is quite possible that in some cases the increase in connective tissue may be secondary to a degeneration of the nerve elements from loss of nutrition or from other causes.

WOUNDS OF THE BRAIN.

The brain may be directly wounded by a foreign body, or indirectly by fragments of bone driven into it, or it may be lacerated by severe contusion without fracture or solution of continuity of the skull. It is very difficult to estimate the degree of injury which must cause death, since persons frequently die from slight, and may recover from very severe, wounds of the brain. In incised wounds of the brain, more or less hæmorrhage occurs at the seat of lesion, and the brain tissue in the vicinity soon undergoes degenerative changes. These may be comparatively slight or extensive. Inflammatory reaction may occur in the vicinity, and the adjacent brain tissue, as well as the hæmorrhagic and degenerated area, become infiltrated with pus cells. After a time the injured and degenerated area may become surrounded by new-formed connective tissue, and the decomposed extravasated blood and detritus of brain tissue, more or less fatty, may be absorbed, and thus after a time the part heals by a more or less pigmented cicatrix. The healing is in most cases very slow and may occupy months or even years. The pia mater may participate to a marked degree in the inflammatory healing process. Abscesses may form near the seat of injury.

After wounds which involve the removal of portions of the cranial bones, it is not uncommon after a few days to see a bleeding fungous mass project through the opening. This mass, sometimes wrongly called hernia cerebri, consists of degenerated brain tissue, blood, and granulation tissue, with more or less pus. The brain tissue below it is degenerated, broken down, soft, and purulent, and there is often abscess in the adjacent brain tissue. Such wounds may finally heal by the absorption of the broken-down brain tissue and blood, and its substitution by granulation tissue.

Lacerations of the brain tissue without fracture may appear shortly after the injury as simple more or less circumscribed areas of capillary hæmorrhage; the brain tissue about these may degenerate, pus may form, and abscesses be developed; or the degenerated and lacerated tissue may be gradually replaced by granulation tissue which finally forms a cicatrix. The process of degeneration and softening and of healing in such lacerations of brain tissue may occur very slowly indeed, even occupying years, and not infrequently the degenerative changes are very extensive and progressive. In many cases, of course, the injury is so extensive, or involves such important parts of the organ, that very little or no inflammatory or degenerative change takes place before the occurrence of death.

Encephalitis in the New-born.—This condition, first described by Virchow, is said to consist in the formation of circumscribed collections of cells of various sizes containing many fat-granules (granular corpuscles) and forming yellowish masses, from 1 mm. to 6 mm. in diameter, in the brain tissue. A more diffuse occurrence of granular corpuscles is also described, but this is said by some observers to be physiological. The nature of this lesion is but little understood, and is still the subject of controversy.

Holes or Cysts in the Brain.—Larger and smaller holes may be found in the brain tissue from dilatation of the perivascular lymph spaces, or well-formed cysts may exist from hæmorrhage, inflammatory softening, hydatids, etc. There are, however, cases in which one or several holes of varying size are found in the brain which cannot be determined to have either of the above modes of origin. They may lie deep in the brain substance or close under the pia mater, or may communicate with the ventricles. This condition is sometimes called *porencephalie*, and may co-exist with various mental aberrations, hydrocephalus, etc.[1]

Syphilitic Inflammation of the Brain sometimes results in the formation of so-called gummy tumors. These are most frequently found near the periphery of the brain, not infrequently connected with the meninges, and may be sharply circumscribed. The central portion of the tumor is usually in a condition of cheesy degeneration, and in the periphery we see fibrous tissue or a dense infiltration of small spheroidal cells.

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[1] Consult *Kundrat*, "Die Porencephalie," Graz, 1882, and *Savage* and *White*, "On the Causes of Holes in the Brain," Trans. London Path. Soc., Vol. 34, page 1, 1883.

Syphilitic inflammation of the brain very frequently occurs in a diffuse form, characterized by the formation of a gelatinous grayish tissue consisting of a more or less homogeneous or granular basement substance, with numerous small spheroidal cells. The nerve elements are atrophied. Obliterating endarteritis may occur as a result of syphilitic poisoning (Fig. 95).

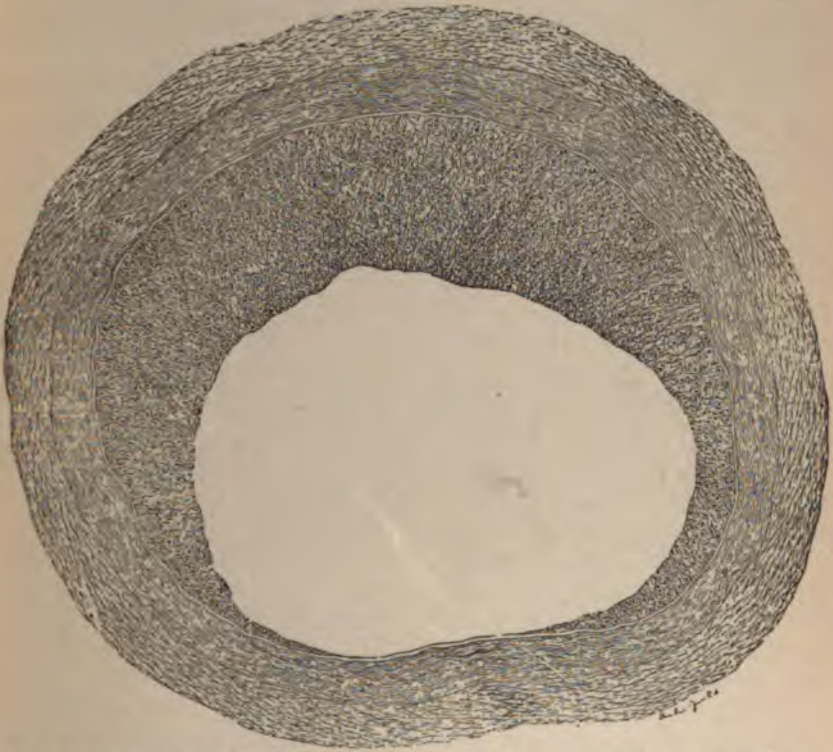

FIG. 95.—SYPHILITIC OBLITERATING ENDARTERITIS OF A CEREBRAL ARTERY, × 50 and reduced.

Tubercular Inflammation of the brain substance usually manifests itself in the formation of circumscribed masses of new tissue from 5 mm. to 6 mm. in diameter, or larger. These may be single or multiple, are most common in young persons, and very frequently occur in the cerebellum. They are apt to occur in connection with tubercular inflammation of other organs. They are frequently called *solitary tubercles*, and usually consist of a dense central cheesy mass, around which is a grayish zone containing tubercle granula, numerous small spheroidal cells, with occasionally larger polyhedral cells and giant cells (Fig. 96). They do not, as a rule, seem to be formed by an aggregation of miliary tubercles, although these may be present in the periphery. Tubercle bacilli have been found in these solitary tubercles.

They sometimes suppurate and break down, and then may simulate simple abscesses.

Conglomerate and scattered miliary tubercles of the ordinary form sometimes occur in the brain, usually in connection with tubercular inflammation of the meninges or ependyma.

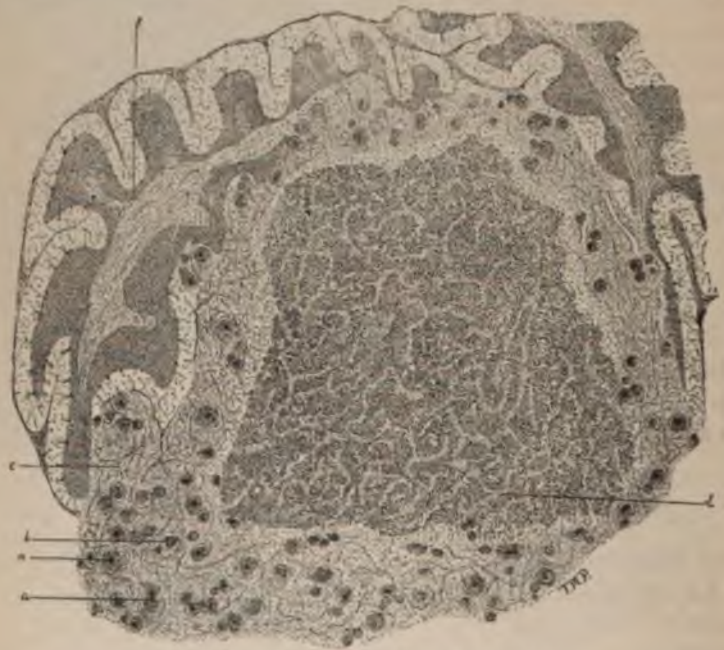

FIG. 96.—SOLITARY TUBERCLE OF CEREBELLUM.

a, *a*, miliary tubercles with giant cells; *b*, *b*, miliary tubercles without giant cells; *c*, diffuse tubercle tissue; *d*, central cheesy mass; *e*, nerve tissue of the cerebellum.

LESIONS OF THE BRAIN IN GENERAL PARESIS OF THE INSANE.

The changes in this disease are in the main those of chronic diffuse encephalitis, but the appearances vary greatly and depend to some extent upon whether the brain is examined in early or late stages of the disease. According to Meyer, in the early stages of the disease, the convolutions, particularly of the anterior cerebral lobes, are swollen, the gray matter congested and softened in places. The brain tissue is more or less infiltrated with leucocytes. Fatty degeneration of the walls of the capillaries, and punctate hæmorrhages, are also common.

In later stages of the disease, a great variety of changes may be observed: hæmorrhagic pachymeningitis, thickening of the dura mater, and close adhesions to the skull; thickening and opacities of the pia mater, adhesions of the latter to the dura mater and to the brain tissue. The brain tissue is apt to be atrophied, and the ventricles dilated and filled with fluid. The pia mater may be œdematous, the ependyma thickened and roughened. On microscopical examination, the neuroglia is found to be increased in amount, the ganglion cells shrunken and sometimes pigmented; the nerve fibres may also be atrophied, and the blood-vessels in a condition of fatty or hyaline degeneration. There may be an accumulation of fatty and granular cells along the walls of the blood-vessels. Secondary degenerations in the spinal cord are not infrequently observed.

It is very difficult to make positive and definite statements regarding many such lesions of the brain as those just indicated, or in general of brain lesions whose nature must be revealed by microscopical study, because our technical procedures in the study of the brain, even in normal conditions, are still quite unsatisfactory and incomplete. The brain tissue is so delicate and liable to post-mortem changes, and the effects of different preservative agents are so liable to variations, that great caution is necessary in arriving at conclusions regarding the minuter lesions affecting the nerve tissue of the brain.

HYPERTROPHY AND ATROPHY OF THE BRAIN.

True Hypertrophy of the brain is rare, and probably always congenital. An increase in the size of the brain from the proliferation of the neuroglia sometimes occurs in children either before or after birth, less frequently in youth, and very seldom in adults. The white substance of the hemispheres is increased in amount. If it takes place before the ossification of the cranium, the bones are separated at the sutures and fontanelles; if after this, the inner table of the skull may be eroded and thinned. When the cranium is opened, the dura mater appears tense and anæmic, the convolutions of the brain are flattened, the brain substance is firm and anæmic, the ventricles are small, the ganglia and cerebellum are either of normal size or compressed.

The disease is usually very chronic, and destroys life with symptoms of compression of the brain. There may, however, be acute exacerbations.

Atrophy.—This may occur as a senile change, or, in adults, in chronic alcohol, opium, or lead poisoning, in chronic insanity, and in chronic meningitis. In children who are much reduced by chronic diseases, atrophy of the brain may accompany atrophy of the rest of the body.

The atrophy affects principally the cerebral hemispheres, and may be uniform or more marked in some parts than in others. The convolutions are small, the sulci broad, the ventricles usually dilated, the brain tissue firm, the gray matter discolored, the white substance grayish in color; the blood-vessels may be dilated. The basal ganglia may be small. Serum accumulates in the pia mater and the ventricles; the pia mater, and often the skull, become thickened; the brain tissue may be œdematous or contain small hæmorrhages. The nerve elements of the brain tissue are those most involved in the atrophy.

Pigmentation.—This may occur in any portion of the brain or its meninges from the decomposition of extravasated blood. In persons affected by malaria, the gray matter of the brain has sometimes an unusually dark or even blackish appearance. This color is due to the presence of black pigment granules within the capillary blood-vessels. The obstruction to the vessels by masses of these pigment granules may cause capillary apoplexies. The pigment may also be found in the walls, and in the lumina of the vessels of the pia mater.

Pigmented patches of congenital origin are not infrequently seen in the pia mater. They may be due to the presence of branching pigmented cells.

TUMORS OF THE BRAIN.

The most common tumors of the brain are *gliomata*. They are most frequently found in the cerebrum, and may be small or large. They may be sharply circumscribed or merge imperceptibly into the brain substance. They may be white and hard, or gray and soft or gelatinous. The softer forms consist largely of neuroglia cells (Fig. 61), the harder forms contain fewer cells and numerous interlacing fibrils (Fig. 60). The blood-vessels of the gliomata are often widely dilated. They are frequently the seat of hæmorrhages and may soften and break down, and may then resemble apoplectic clots. They are liable to undergo fatty and cheesy degeneration.

Not infrequently the gliomata are combined with sarcomata, forming *glio-sarcomata*. Sarcomata of various forms occur in the brain as primary tumors.

Circumscribed masses of dilated blood-vessels, *angiomata*, are not infrequently met with in the brain. *Myxoma*, *fibroma*, *lipoma*, and *osteoma* are among the rarer forms of brain tumors. *Endotheliomata* (*cholesteatomata*), in the form of glistening white tumors, sometimes attain a large size. *Sarcomata* and *carcinomata* not infrequently occur in the brain as secondary tumors. *Dermoid* and other *cysts* occur, but are rare.

Small masses of the gray matter of the brain sometimes occur in the medullary portions of the brain, and localized hyperplasias of gray matter have been described in the form of rounded nodules projecting from the free surface of the brain or into the ventricles.

PARASITES.

Cysticercus and, more rarely, echinococci are found in the brain.

MALFORMATIONS.

Cyclopia.—This malformation consists in an arrest of development affecting the cerebrum, which, instead of separating into two hemispheres, remains single, with one ventricle, and the rudiments of the eyes usually become joined and form one eye. This single eye is in the middle of the face, near the place of the root of the nose, in a single orbit. Over this is an irregular body representing the nose. The rest of the face is well formed. Or the eyeball may be wanting entirely, or there are two eyes joined together. or, more seldom, two separate eyes. The orbit is surrounded by rudiments of four eyelids. The frontal bone is single, the nasal bones undeveloped; the ethmoid, vomer, and turbinated bones are absent. The optic nerve is double, single, or absent. There may be hydrocephalus. Such children are incapable of prolonged existence.

Anencephalia.—This malformation may be of various degrees. The brain may be entirely absent, and the base of the cranium is covered with a thick membrane, into which the nerves pass. Or the membranes may form a sort of cyst containing blood and serum, or portions of brain. Of the cranial bones, only those which form the base of the skull are present (*Acrania*). The scalp is usually partly or entirely absent over the opening in the skull; the eyes stand prominently out, and the forehead slopes sharply backward. This malformation may occur in otherwise well-developed children.

Hydrocephalus.—This lesion has been already considered on p. 179. It is probable that in some cases *hydrocephalus internus* is due to a primary *partial anencephalia*, and that the accumulation of fluid is of secondary occurrence. In rare cases, only part of one lateral ventricle is hydrocephalic, giving to the head a protuberance on one side. The viability of the fœtus depends upon the degree of the hydrocephalus. *Hydrocephalus externus* is an accumulation of serum beneath the pia mater, or, according to some authors, between the pia and dura mater. It causes dilatation of the cranium and compression of the brain. It is of very rare occurrence, and may also be secondary to partial anencephalia.

Cephalocele, or Brain Hernia.—When abnormal openings exist in the skull from malformation, the contents of the cerebral cavity are apt to protrude in the form of larger or smaller sacs. This may occur in cases of well-marked anencephalia, or in cases in which the brain is well developed. The protruding sac formed of the meninges may or may not be covered with skin. If the contents of the sac are simply fluid, the lesion is called *hydromeningocele;* if composed of brain substance, *encephalocele;* if the sac contain both fluid and brain substance, it is called *hydrencephalocele.* The sacs may be very small or as large as a child's head. They may protrude from the top of the skull in acrania. They most frequently protrude through openings in the occipital bone, often hanging down in large sacs upon the neck; also at the root of the nose, along the line of the sutures, at the base of the skull, and elsewhere.

Microcephalia.—This is an abnormally small size of the brain, with a correspondingly small cranium. The diminution in size affects principally the cerebral hemispheres, though the other parts of the brain are also small. The convolutions are few and simple, the cavities often dilated with serum; on the membranes there may be traces of inflammation. The cranium is small, the face large, the rest of the body small. The malformation is in some cases caused by inflammation or dropsy of the brain during fœtal life. It is endemic in some countries, but single cases may occur anywhere. The fœtus is viable. Absence or incomplete development of portions of the brain may occur, not only in idiots, but in persons whose minds are perfect.

THE SPINAL CORD.

THE MEMBRANES OF THE SPINAL CORD.

A.—THE DURA MATER SPINALIS.

The dura mater spinalis, unlike that of the brain, does not serve as periosteum to the bones forming the cavity, so that the lesions of the two membranes differ somewhat. *Hæmorrhage* may occur, as the result of injury, between the dura mater and periosteum, or it may occur in tetanus, as a result of circulatory changes induced by muscular spasm, or in the asphyxia of new-born children. Small hæmorrhages on the surfaces of the membrane may occur as the result of inflammation.

Serous fluid may accumulate outside of the dura mater as a result of post-mortem changes, or in connection with circulatory or inflammatory changes in the membranes.

Inflammation.—Acute external pachymeningitis is almost always secondary to disease or injury of the spinal column, and may result in collections of pus between the dura mater and periosteum, usually most abundant posteriorly. *Hæmorrhagic pachymeningitis* occurs in the dura mater spinalis, with the formation of products similar to those observed in the brain, in the chronic insane and in drunkards. *Simple chronic pachymeningitis interna,* with the formation of new connective tissue containing brain sand, is not infrequent. The new tissue may form minute projections or roughness of the surface, or, when more abundant, the *psammomata*. *Tubercular inflammation* of the dura mater spinalis may occur in connection with tubercular meningitis, or secondary to tubercular inflammation of the vertebræ.

Tumors.—Fibromata, lipomata, chondromata, myxomata, endotheliomata, and *adeno-sarcomata*[1] occur in the dura mater spinalis as primary tumors. *Carcinomata* and *sarcomata* may occur as secondary tumors. Small plates of new-formed *bone* are rarely found in the dura mater spinalis.

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[1] *Hodenpyl:* American Journal of the Medical Sciences, March, 1888.

Parasites.—Echinococcus developing outside of the spinal canal may perforate the dura mater; or the cysts may lie between the dura mater and the pia mater.

It is obvious that even small tumors in the spinal canal may give rise to serious results from compression.

B.—THE PIA MATER SPINALIS.

It is almost impossible in most cases in the pia mater, as well as in the dura mater spinalis and in the spinal cord, to judge with certainty, from the appearances after death of the blood-contents of the vessels, of these parts during life. The same is true of abnormal quantities of serum found after death. The veins of the pia mater, especially in the posterior region, may be greatly distended with blood after death, without pre-existing disease; and the intermeningeal space may contain much fluid under the same condition.

Hæmorrhages may occur from injury in connection with severe convulsions, or general diseases such as the hæmorrhagic diathesis, scurvy, small-pox, etc. The hæmorrhages under these conditions, except from injury, are not usually extensive. But in some cases of injury, or cerebral apoplexy; from the bursting of aneurisms of the basilar or vertebral arteries; or in cases in which we cannot find a cause, a very large quantity of blood may collect between the dura and pia mater, and in the meshes of or beneath the latter.

Inflammation.—Acute exudative spinal meningitis occurs under essentially the same conditions and with essentially the same post-mortem appearances as acute cerebral meningitis, though it is less frequent. The exudations are apt to be most abundant in the posterior portions. It may be associated with a similar inflammation of the pia mater cerebralis, and the inner surface of the dura mater may be involved. The disease may be circumscribed, but usually affects the entire length of the membrane.

Tubercular inflammation is usually most marked, when associated with a similar condition of the pia mater cerebralis, in the upper portions of the cord; but it may extend over the entire membrane. The conditions under which it occurs and the character of the lesions are similar in both. *Chronic spinal meningitis* is not infrequent, manifesting itself in the formation of larger or smaller patches of new connective tissue or thickenings of the pia mater. The pia and dura mater may thus be firmly united in places by adhesions, or the pia mater may become closely adherent to the substance of the cord.

Not very infrequently large numbers of pigment cells are found in the pia mater spinalis, sometimes giving it a distinct gray or blackish color.

Tumors.—Small plates of *cartilage* and *bone* are sometimes found in the pia mater.

Fibromata, myxomata, sarcomata, and *endotheliomata* have been found.

Parasites.—Cysticercus sometimes occurs in the meshes of the pia mater.

THE SPINAL CORD.

Hæmorrhage.—This is much less frequent than in the brain, but may occur either as *capillary apoplexy* or as larger *apoplectic clots.* Capillary hæmorrhages, similar in appearance to those of the brain, may occur as the result of injury, or near areas of softening or tumors, or may accompany severe convulsions, as in tetanus. Apoplectic clots, which are comparatively rare in the spinal cord, are usually small, commonly not more than one cm. in diameter, and are similar in their appearances, and in the changes subsequent to their formation, to those of the brain. They are usually the result of injury; but may occur spontaneously, probably in most cases as a result of inflammation, and are then most apt to occur in the gray matter. Sometimes, however, hæmorrhagic foci are found in the spinal cord without traumatism or evidence of inflammatory change.

INJURIES.

The spinal cord may be compressed or lacerated, by penetrating wounds, by fracture or dislocation of the vertebræ, or by concussion without injury to the vertebræ. The spinal cord is found simply disintegrated, or there may be much hæmorrhage and the disintegrated nerve tissue be mixed with blood. If life continue, the nerve elements may degenerate, Gluge's corpuscles and free fat-droplets may form; blood-pigment may be formed, and when inflammation supervenes, more or less pus may be intermingled with the degenerated detritus. There may be marked changes in the minute structure of the cord without any change being evident to the naked eye.

SECONDARY DEGENERATIONS IN THE SPINAL CORD.

When nerve fibres of certain parts of the brain and of the spinal cord are divided or destroyed from any cause, that portion of them which becomes separated from its trophic centres degenerates. After a time—frequently two to four weeks—the medullary sheath and axis cylinder disintegrate, becoming granular and fatty. These products of degeneration may be in part absorbed at once, or may collect in cells, forming the so-called compound granular corpuscles. After a still longer time—sometimes several months—the degenerated areas become gray in color, from the absorption of the degenerated myelin, harder, and somewhat shrunken. These changes are partly due to the formation of new connective tissue which takes the place of the degenerated nerve fibres.

Since the affected portion of nerve tissue becomes gray or translucent after the myelin is broken down and absorbed, and the new connective tissue is formed, this is often called *Gray Degeneration ;* or as the degenerated areas are harder than normal, it is sometimes termed *Sclerosis.*

Now, it is found that this secondary degeneration takes place in the direction in which the fibres conduct—in centripetal or sensory fibres, upward ; in centrifugal or motor fibres, downward. Thus, we have *Descending Gray Degeneration* (*Descending Sclerosis*), and *Ascending Gray Degeneration* (*Ascending Sclerosis*).

Descending Gray Degeneration.—This change affects only the motor nerve fibres, and may reach but a short distance from the seat of lesion, or may extend for a long distance, depending upon whether the severed fibres run a short or long course before reaching their termination, *i.e.*, passing into ganglionic centres. Lesions of the brain, such as embolic softenings and apoplectic clots, which destroy or interrupt any of the motor nerve fibres originating in the central convolutions, may be followed by gray degeneration of the portion of the fibres situated peripherally to the lesion. These fibres pass through the corona radiata, anterior portion of the internal capsule, pes pedunculi, pons, and thence to the anterior pyramids, where most of them decussate and pass to the posterior part of the lateral columns of the opposite side. Those which do not decussate form a narrow band at the inner part of the anterior columns of the same side, constituting the *columns of Türck.* These fibres which convey motor impulses from the brain to the cord form a system called the *pyramidal tract.* There are also short motor fibres in the anterior columns, forming the so-called *anterior root zones,*

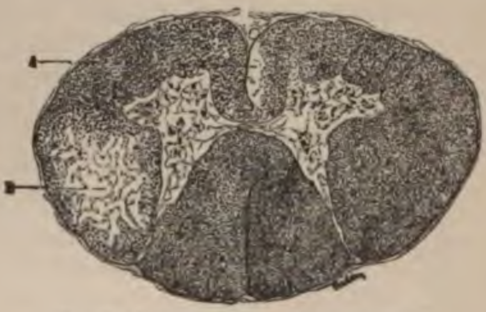


FIG. 97.—DESCENDING GRAY DEGENERATION.

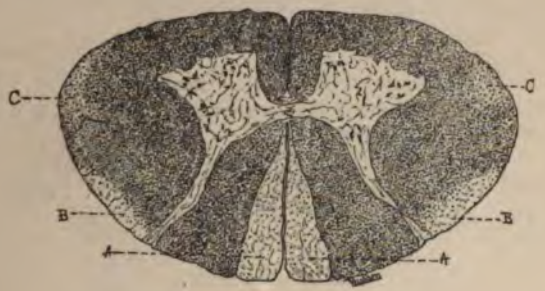
Section of cord in cervical region, showing secondary degeneration from softening in pons which had existed for at least six months. A, column of Türck on same side as brain lesion; B, pyramidal tract on opposite side.

which maintain communications between different portions of the gray matter.

Now, a lesion in the brain or medulla, destroying the continuity of the motor nerve fibres of the pyramidal tract, will be followed by areas of gray degeneration in the posterior part of the lateral column of the opposite side, and in a narrow band near the anterior longitudinal fissure of the same side (see Fig. 97). A lesion below the medulla, involving the fibres of the pyramidal tract, will be followed by degeneration of the fibres on the same side below the point of lesion. Lesions involving the fibres of the anterior root zones will be followed by degenerations which extend but a short distance, since these fibres soon communicate with their centres in the gray matter. If a part only of the fibres in any of these regions are interrupted, the amount of degeneration is proportionately small.

Ascending Gray Degeneration.—Any lesion interrupting the course of the sensory nerve fibres in the cord is followed by degeneration of the central ends of the involved fibres, and increase of connective tissue about them. These fibres are in part situated in the posterior columns, and form communications between different parts of the gray matter—*posterior root zones*—and hence have not great length. Other sensory fibres are grouped in a narrow band near the posterior longitudinal fissure, forming the *columns of Goll*, while other sets, forming the so-called *direct cerebellar tract* and the columns of Gowers, are situated in the periphery of the lateral columns.

P. H. Hatters, M.D.



P. H. Hatters, M.D.

FIG. 98.—ASCENDING GRAY DEGENERATION.

Section of cord in cervical region, showing secondary degeneration from a tumor in the dorsal region of 18 to 20 months' duration. A, columns of Goll; B, pyramidal tract; C, columns of Gowers (antero-lateral ascending tract). Drawn from a specimen prepared by Dr. Ira Van Gieson.

A lesion of the cord involving the severance or destruction of these centripetal fibres will be followed by gray degeneration of the entire posterior columns, and the cerebellar tract, and the columns of Gowers for a short distance above the lesion. The fibres of the posterior root zone being short, however, the degeneration will, at a short distance above the lesion, become limited to the columns of Goll, the direct cerebellar tract, and the columns of Gowers in the lateral columns (Fig. 98). The degeneration may be traced along the columns of Goll to the restiform bodies, and in the cerebellar tract to the cerebellum. Lesions involving the entire thickness of the cord will produce bilateral degenerations.

It should be borne in mind, in looking for these secondary lesions, that they are not developed until considerable time has elapsed since the primary lesion, and that, when small areas are involved, they are usually inconspicuous. In any event, the lesions are apt to be more evident to the naked eye in specimens hardened in chromic fluids than when fresh, and microscopical examination is often necessary for their recognition (Fig. 99).

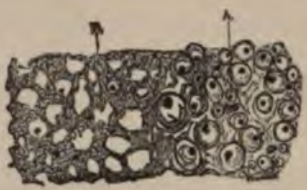


FIG. 99.—ASCENDING GRAY DEGENERATION.

A small portion from edge of degenerated region of cord shown in Fig. 98, more highly magnified. A, normal nerve fibres; B, degenerated area.

INFLAMMATION.

Acute Myelitis.

This lesion of the spinal cord, which is sometimes distinctly inflammatory in character, and sometimes of a rather degenerative nature, is usually confined to a comparatively limited longitudinal extent of the cord, and hence is sometimes called *transverse myelitis.* When the cord is removed and laid upon the table, if the lesion is marked a flattening of the cord at its seat may be observed; or on passing the finger gently along the organ, the affected segment will be found softer than the rest of the cord. On making a section through the diseased portion, the nerve tissue may be white or red or yellowish or grayish; it may be quite firm, but is usually more or less softened and sometimes almost diffluent.[1]

Microscopical examination shows different appearances, depending upon the stage of the inflammatory or degenerative process. There may be much blood, or, if the lesion has existed for some time, blood pigment; also fragments of more or less degenerated nerve fibres and cells (Fig. 100), myelin droplets, free fat-granules, and larger and smaller cells filled with fat-granules (Gluge's corpuscles), pus cells, granular matter, neuroglia cells, and sometimes corpora amylacea. The various combinations of these elements give rise to the different gross appearances which the diseased part presents. In earlier stages of the lesion, the blood-vessels may be dilated, the nerve fibres and cells swollen; or the walls of the blood-vessels may be thickened or fatty.

---

[1] It should be remembered that a mechanical injury to the cord in removal, such as crushing or bruising, may reduce the injured portion to a pulpy consistency and thus produce appearances somewhat similar to those of some forms of inflammatory softening.

The lesion is apt to commence in the gray matter or at its edge, and then extend first laterally and afterwards upward and downward.

In a certain number of cases, the degenerated material may be absorbed and a cicatrix or cyst formed. In the least extensive forms of the lesion, there is apparently a regeneration of the nerve fibres and a restoration of the functions of the cord.

P. K. W.

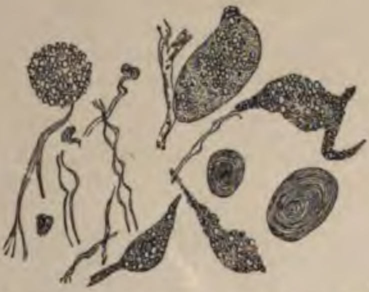


FIG. 100.—DEGENERATED TISSUE FROM ACUTE MYELITIS.

Secondary gray degeneration, both ascending and descending, may occur in this form of myelitis, varying in extent according to the size of the primary lesion.

Acute disseminated Myelitis runs a rapid course, and proves fatal in a short time. The inflammation involves nearly the whole length of the cord, but is more intense in some places than in others. The cord is swollen and congested, it is infiltrated with pus cells, the connective-tissue framework is swollen, and the nerve elements are degenerated.

Poliomyelitis anterior (*Myelitis of the anterior horns*).—This name is applied to a group of cases which are characterized by clinical symptoms indicating changes in the anterior gray cornua. The disease occurs both in children and in adults, and varies in the severity, acuteness, and duration of its symptoms. In many cases, there is complete recovery, and then we must suppose that the changes in the nervous tissue were not destructive in their character. In other cases, the symptoms ar more permanent, indicating a destructive lesion. From the autopsie so far recorded, we learn that the lesion is most frequent at the lumbar and cervical enlargements of the cord, but may occur anywhere, and is often in scattered patches. There is degeneration, shrinkage, pigmentation, and atrophy of the ganglion cells in the anterior gray cornua (Fig. 101). There may be an increase of connective tissue in the gray cornua and in the anterior and lateral columns. There may be degene-

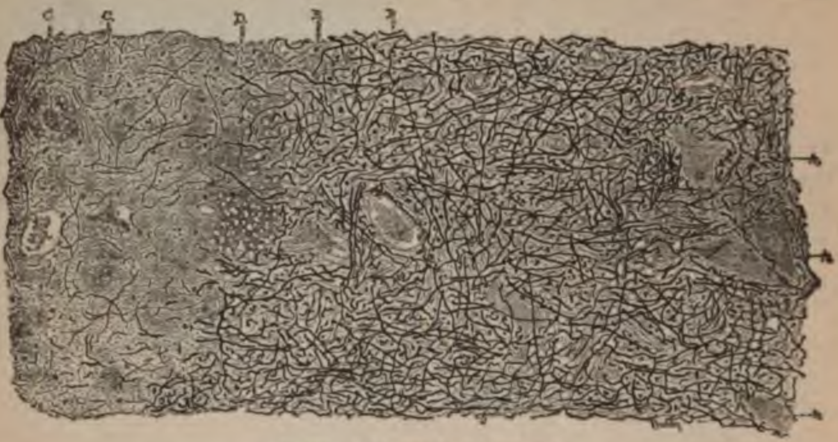


FIG. 101.—POLIOMYELITIS ANTERIOR.

Showing portion of Fig. 102 at edge of affected area, more highly magnified. A, normal ganglion cells surrounded by nerve fibres; B, degenerated ganglion cells; C, granular masses at place of ganglion cells; D, small cavity containing fluid.

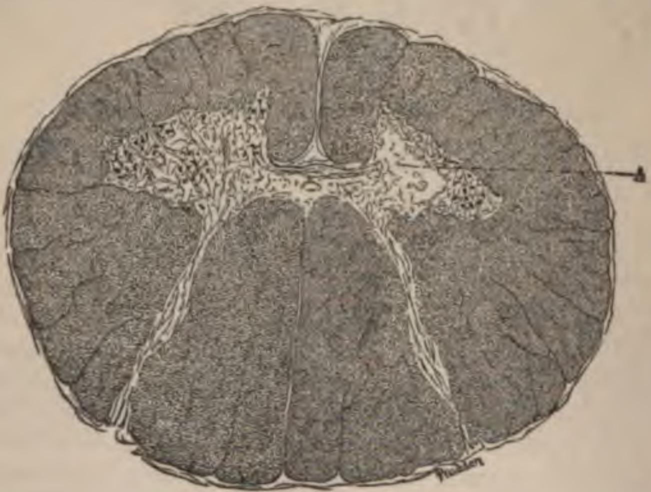


FIG. 102.—POLIOMYELITIS ANTERIOR.

Showing degenerated area in anterior cornua, with atrophy of gray matter. A, atrophic region (Weigert's hæmatoxylin stain). Specimen prepared by Dr. Ira Van Gieson.

ration and destruction of a considerable part of the anterior cornua; there may be atrophy of the anterior nerve roots. The cord may be considerably distorted as the result of the lesion (Fig. 102).

Chronic Myelitis.

Chronic Interstitial Myelitis.—Under this heading are embraced a variety of lesions which probably differ from one another somewhat in the nature of the changes involved, but more in the seat of the disease. We shall consider without special classification the most important forms.

Chronic Transverse Myelitis.—In certain cases of pressure on the spinal cord from a tumor or from displacement of the bones of the vertebral column, etc., instead of becoming softened or undergoing acute inflammatory changes, the cord becomes the seat of a localized formation of new connective tissue, with consecutive atrophy of more or less of the nerve elements in the gray and white matter. The cord becomes in this way harder, and sometimes shrunken at the seat of lesion, and grayish in color. This change may be followed by ascending and descending gray degeneration.

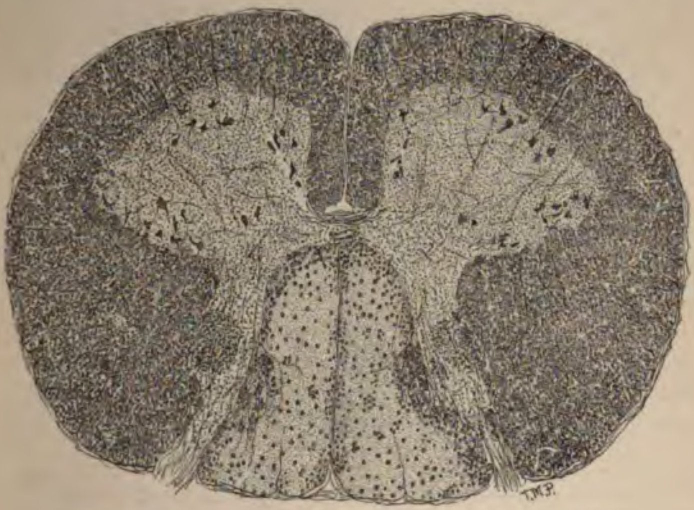


FIG. 103.—POSTERIOR SPINAL SCLEROSIS. Upper dorsal region.

Multiple Sclerosis.—This lesion, similar in its nature to multiple sclerosis of the brain, and often occurring with it, seems to belong, like other chronic inflammations of the cord considered in this section, to the group of chronic interstitial inflammations. It consists in the formation, in more or less numerous, scattered, circumscribed areas, of new connective tissue, apparently derived from the neuroglia. The formation of new connective tissue is accompanied by degeneration and atrophy of the nerve fibres and ganglion cells. Whether the formation of new connective tissue is primary and the atrophy of the nerve elements secondary, or *vice versa,* it is difficult to say. The new connective tissue consists of the characteristic branching neuroglia cells surrounded by a more or less dense network of fine fibrillæ, many if not most of which seem to be branches of the neuroglia cells. Corpora amylacea and sometimes fat-droplets, either free or contained in cells, may be present in the sclerosed areas.

The areas of sclerosis may involve both gray and white matter, and may be very small or large. If very small, or in early stages of formation, they may not be recognizable by the naked eye, but when visible they are grayish, translucent, and firmer than the surrounding tissue, and may or may not present a depressed surface; they sometimes project above the general level. The cause of this, as of other forms of so-called idiopathic interstitial myelitis, is very obscure.

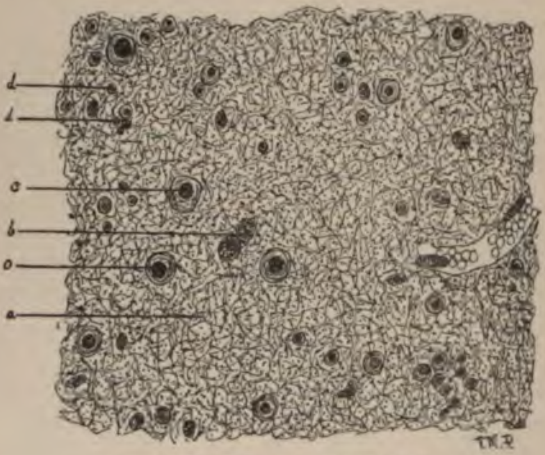


FIG. 104.—POSTERIOR SPINAL SCLEROSIS.

A portion of sclerosed area shown in Fig. 103, more highly magnified. *a*, new-formed connective tissue; *b*, blood-vessels; *c*, nerve fibres; *d*, atrophied nerve fibres.

Posterior Spinal Sclerosis (*Locomotor Ataxia*).—This lesion consists essentially in a degeneration and atrophy of the nerve fibres and an increase of connective tissue in the posterior columns of the spinal cord (Fig. 103). Not infrequently the posterior portion of the lateral columns, the posterior roots and cornua are also involved. Exceptionally a large part of the lateral columns is involved, and also the anterior cornua. The change usually commences in that portion of the posterior columns bordering on the posterior cornua, but may involve, as above stated, the adjacent parts. It is usually most marked in the lower dorsal and lumbar regions. The sclerosis may extend upward to the restiform bodies, but in the cervical region it is apt to be confined largely to the columns of Goll, although there are exceptions to this.

When the lesion is well developed, the pia mater over the affected area is usually thickened and adherent to the cord. In its early stages there may be no change evident to the naked eye; but when advanced, the posterior columns may appear somewhat depressed, and grayish and firmer than the rest of the cord. The microscopical appearances vary, depending upon the stage and extent of the lesion. The walls of the blood-vessels may be thickened; there is more or less new connective tissue consisting of neuroglia cells and very numerous interlacing, delicate fibrils. There may be numerous corpora amylacea and fat-granules, either free or collected in cells. The nerve fibres may be numerous, but separated more or less widely by the new connective tissue, or they may be very few in number and irregularly scattered through the new tissue (Fig. 104). The atrophy may involve the fibres of the posterior nerve roots and cornua, and even the ganglion cells of the latter.

According to the recent researches of Lisauer,[1] the columns of Clarke in the dorsal region show in this disease a very constant and marked diminution in the number of delicate fibrils which under normal conditions surround the ganglion cells.

In the rare cases in which the sclerosis extends to the lateral columns and to the anterior cornua, the minute characters of the lesions are the same.

There are other less important and less well understood forms of sclerosis of the white matter and degenerative or inflammatory changes in the gray matter of the cord; but, so far as we know, the character of the lesions is essentially the same as those above described, except in their distribution. They are associated either with paralysis or changes in the muscles, and the secondary changes are for the most part better known than the primary alterations in the cord. Such lesions, which the scope of this book does not permit us to enter into, are *lateral sclerosis*, *peripheral sclerosis*, the lesions involving *muscular atrophy*, etc.

Solitary tubercles and *gummata* may occur in the spinal cord, but are not common. *Cysts* may occur as a result of softening, or from unknown causes. Sometimes very long, narrow canals are found in the spinal cord, even reaching nearly its whole length. Some of these are evidently the dilated central canal, as they are lined with epithelium. Others, however, have been found behind the central canal, and their mode of origin is unknown.

Tumors.—Tumors of the spinal cord are not very common, much less so than tumors of the meninges. *Fibromata, gliomata, sarcomata, glio-sarcomata.* and *neuromata* have been described as primary tumors. *Carcinomata*, as secondary tumors, may occur. Tumors of the meninges are apt to secondarily involve the cord.

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[1] Fortschritte der Medicin, Bd. ii., No. 4, 1884.

SYRINGOMYELIA.

This lesion of the spinal cord consists of the formation of gliomatous or glioma-sarcomatous tissue in the vicinity of the central canal, and its subsequent partial disintegration with the formation of one or more cavities within the substance of the cord (Fig. 105). These cavities, which are filled with fluid, vary greatly in size, shape, and extent, and while usually situated in the central region of the cord, they may involve the anterior and posterior cornua and invade the posterior columns. There may be two communicating cavities, and these may, but usually do not, open into the central canal. The longitudinal extent of these cavities varies greatly. The lower cervical and upper dorsal regions are most frequently involved. The cavity is usually lined with tissue somewhat denser than that which makes up the bulk of the tumor. The gliomatous or glio-sarcomatous tissue, which forms the basis of the lesion in syringomyelia, probably originates from the layer of neuroglia which surrounds or extends away from the central canal.

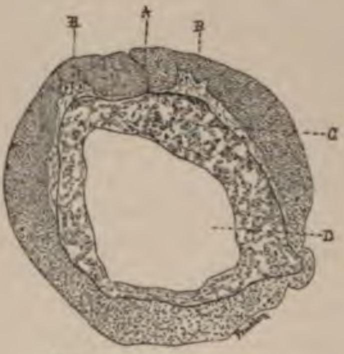

FIG. 105.—SYRINGOMYELIA.

Transverse section of cord. A, white substance of cord, distended by tumor; B, B, distorted and atrophied gray substance of anterior cornua; C, tumor mass (glio-sarcoma); D, cavity in cord. Drawn from specimen prepared by Dr. Van Gieson.

Syringomyelia is frequently mistaken for hydromyelia (see below), which is a congenital malformation, and in which the longitudinal cavity in the cord is at some period lined with epithelial cells.

There seems, furthermore, to be a class of lesions of the cord, usually classed as syringomyelia, in which cavities of various forms coexist with a tumor in the vicinity of the central canal. But these cavities do not appear to be formed by a breaking-down of the tumor tissue, but in some other way as yet little understood.

Altogether the limitations of the term syringomyelia are somewhat indefinite, and will no doubt remain so until we possess fuller knowledge of the genesis and structure of the neuroglia tissue of the spinal cord.

MALFORMATIONS.

The spinal cord may be double either in circumscribed portions or over a large part of its extent. It may be unusually long or short, or it may be absent altogether (*amyelia*). This usually occurs with anencephalia. Under these conditions a long connective-tissue sac, filled with more or less fluid, may occupy the spinal canal.

Hydromyelia (*Hydrorrhachis interna*).—This abnormality consists in the dilatation of the central canal of the spinal cord by fluid (Fig. 106).

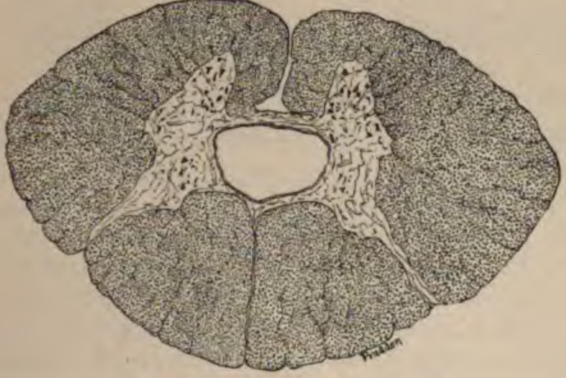

FIG. 106.—HYDROMYELIA.

In the section from which this drawing was made, the epithelial cells surrounding the dilated central canal were well preserved.

This may be moderate, or so extreme that but little of the substance of the cord is left as a thin shell around the central cavity. When they have not been destroyed by atrophy, epithelial cells may be found lining the cavity.

Hydrorrhachis externa.—In this lesion there is an accumulation of fluid between the meninges of the cord, causing more or less atrophy of the latter.

Hydromyelocele—Spina bifida.—In the majority of cases, hydrorrhachis is accompanied by a more or less complete lack of closure of the spinal canal posteriorly, so that the collections of fluid within may pouch outward through the opening, in the form of a sac. The sac may be covered by skin, or this may be absent, either from the beginning or as a result of thinning and rupture. The walls of the sac may consist of the dura mater and pia mater, or, in cases of hydrorrhachis externa, of the dura mater alone; when both are present, they are usually more or less fused together. Inside of the membranes of the sac there may be a shell of distended nerve tissue of the cord, or the spinal cord may be split posteriorly and the sides crowded sideways; or there may be a rudimentary fragment of the cord suspended in the sac or attached to the walls; or the cord may be but little changed and remain inside the spinal canal. The openings in the spinal canal may be due to the complete or partial absence of the vertebral arches, or more rarely the sac may protrude through openings between the completely formed arches. Spina bifida most frequently occurs in the lumbar and sacral regions, but it may occur in the dorsal or cervical regions; or the canal may be open over its entire length. Very rarely it is open on the anterior surface. The protruding sac may be very small or as large as a child's head. The fluid in the sac is usually clear, but may be turbid from flocculi of degenerated nerve tissue.

THE PERIPHERAL NERVES.

Changes in Nerves after Division.—When nerves are divided or a portion destroyed by injury, the nutrition of certain parts of the fibres is interfered with, apparently because of their separation from their trophic centres, and they suffer degeneration; but after a time, if the conditions be favorable, they may undergo regeneration and restitution of function. The degeneration not only affects the entire severed portion, but it occurs at nearly the same time in all parts. The degeneration consists in the breaking up of the medullary sheaths into variously shaped droplets, and the decomposition of these, with the formation of fat, which may remain for some time either free or inclosed in cells, and finally be absorbed (see Fig. 107). The axis cylinder, too, is, in many cases at least, more or less completely destroyed. The neurilemma and its nuclei do not seem usually to undergo degeneration, but may persist and take part in the regeneration of the nerve when restitution occurs.

After a variable time, if the conditions are favorable, the divided ends of the nerve may be united and a regeneration or new formation of nerves in or about the severed portions may occur, so that the function may be resumed. Considerable time is required, frequently months, for the completion of the regenerative process. Degeneration of the nerves not only follows mechanical injuries, such as incision, crushing or tearing, and compression, as from a tumor or dislocation of the bones, but it may result from disease of the special nerve centres with which the nerves communicate, or from inflammation of the nerves themselves.

Acute Exudative Neuritis.—Primary acute inflammation of the nerves may occur as the result of injury, or it may be secondary to an inflammatory process in its vicinity, although, owing to the dense lamellar sheaths and the special blood-supply, the nerve trunks may escape participation in even very severe inflammatory processes in surrounding tissues. The inflamed nerve may be red and swollen and infiltrated with serum and pus cells. The process may undergo resolution or terminate in gangrene and destruction of the nerve, or it may become chronic and result in the formation of new connective tissue.

Degeneration and regeneration of the nerve fibres, similar to those above described as following division of nerve trunks, may occur in acute neuritis.

Chronic Interstitial Neuritis.—This is essentially a chronic interstitial inflammation resulting in an increase of connective tissue in the nerve sheath and intrafascicular bands. As a result of this, the nerve fibres undergo atrophy from pressure; the medullary sheath, and finally the axis cylinder, being, in more or less of the fibres, partially or completely destroyed.

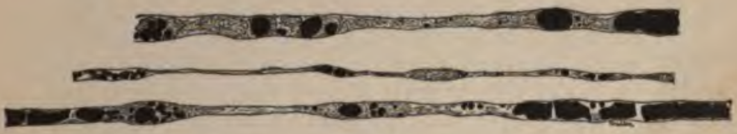

FIG. 107.—MULTIPLE NEURITIS.

From a case of alcoholic poisoning. Specimen stained with osmic acid. The broken-down medullary sheath and fat-droplets are stained deep black.

Multiple Neuritis.—Under a variety of conditions, such as exposure to cold and wet, over-exertion, poisoning by alcohol, arsenic, lead, etc., and in connection with the acute infectious diseases, a degeneration of the nerve fibres in various parts of the body may occur, which may be accompanied with or followed by proliferative changes in the neurilemma cells (Fig. 107). Regeneration of the affected nerve fibres may occur under these conditions, as after experimental division of the nerves, leading to their restitution.[1] In some forms of multiple neuritis, the inflammation is exudative in character, and new cells of various forms are found within and between the nerve fibres. The exact part which the neurilemma and other intrafascicular cells play in the inflammatory and regenerative changes of nerves is not yet very fully made out.

Syphilitic and *Tubercular Inflammation* of the nerves is not common except at their central ends, in connection with similar inflammations of the meninges, or when they are secondarily involved in connection with these inflammations in neighboring tissues.

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[1] Consult *Starr*, "Multiple Neuritis." The Middleton Goldsmith Lecture for 1887. Trans. New York Pathological Society, 1887, page 1.

Leprous Inflammation.—This consists in the formation within the nerve of masses of new-formed tissue somewhat resembling granulation tissue, in whose cells multitudes of characteristic bacilli are uniformly found (see Leprosy). It constitutes the variety of leprosy known as *lepra anæsthetica.*

Tumors.—The tumors of the nerves are such as consist largely of or contain new-formed nerve tissue—*true neuromata;* and the so-called *false neuromata* (Figs. 63 and 64), which are for the most part *fibromata* or *myxomata* of the connective tissue of the nerve. *Myxo-sarcomata* are less common, and *primary sarcomata* rare. The nerves may be secondarily involved in *sarcomata* or *carcinomata,* though not infrequently nerves pass through these tumors without being in the least involved in their peculiar structure.

METHODS OF PREPARATION OF NERVE TISSUE FOR MICROSCOPICAL STUDY.

The general methods of hardening have been already given on page 15. For minute study, there is no one method of staining and mounting upon which we can rely exclusively for the study of all lesions. A preliminary examination of areas of *inflammatory softening,* or of the disintegrated tissue in *apoplectic clots,* or of the new-formed tissue in *chronic hæmorrhagic pachymeningitis interna,* may be made by teasing portions of the affected tissues in one-half-per-cent salt solution. Or the tissues in these lesions, or in any others in which fatty degeneration is suspected, may be placed for twenty-four hours in one-per-cent aqueous solution of osmic acid, and then washed and teased in glycerin. In this way, the myelin and the fat will be stained brown or black. Secondary and other degenerations of medullated nerves may be studied by soaking the nerves for twenty-four hours in one-per-cent solution of osmic acid, and then staining with picro-carmine and teasing and mounting in glycerin. Suppurative inflammation of the central nervous system and the membranes, or the connective-tissue changes in general, may be studied in sections from the tissues hardened in Müller's fluid and alcohol, stained double with hæmatoxylin and eosin (see page 47), and mounted in Canada balsam.

A very useful method of staining sections of nerve tissue, especially of the brain and cord, is that known as Weigert's hæmatoxylin method. The tissue is first well hardened in Müller's fluid.

Blocks of the hardened tissue are imbedded in celloidin, and sections made in the usual way. The sections are first soaked for twenty-four hours in a saturated aqueous solution of neutral cupric acetate diluted with an equal bulk of water. They are now transferred to the hæmatoxylin solution, made as follows:

Hæmatoxylin Crystals	1 gm.
Alcohol, 97 per cent	10 c.c.
Water	90 "
Sat. Aqueous Sol. Lithium Carbonate	1 "

In this solution the sections remain for two hours. (If the finer fibres of the cerebral cortex are to be brought out, the sections must remain for twenty-four hours in the hæmatoxylin solution.) The sections are now thoroughly washed in two or three waters, and then rinsed in alcohol and transferred to the bleaching solution, composed as follows:

Potassium Ferricyanide	2.5 gm.
Sodium Biborate	2. "
Water	200 c.c.

In this fluid the sections discharge a brownish color, and they remain in it until the gray matter has a distinct yellow color and the white matter is bluish black. The time required to produce this effect varies considerably, but is usually from one-half an hour to an hour. The sections are now washed, dehydrated with alcohol, cleared up in oil of cloves or oil of origanum, and mounted in balsam. The sections may be stained in alum carmine before dehydration, to bring out the nuclei.

In sections stained by this method, the gray matter, connective-tissue elements, and ganglion cells have a yellow or yellowish-brown color, the axis cylinders are uncolored or have a slight yellowish tint, while the medullary sheaths are bluish black or black (see Fig. 101).

To demonstrate the presence of miliary aneurisms in or about apoplectic clots, it is usually necessary to macerate the brain tissue in water until the nerve elements disintegrate, and they may then be washed away under a stream of water, leaving the blood-vessels with their aneurisms exposed.

THE RESPIRATORY SYSTEM.

THE LARYNX AND TRACHEA.

MALFORMATIONS.

The larynx and trachea may be entirely absent in acephalic monsters. The larynx may be abnormally large or small. The epiglottis also may be too large or too small, or may be cleft. There may be communications between the trachea and the œsophagus, and then the pharynx generally ends in a cul-de-sac, and the œsophagus opens into the trachea. There may be imperfect closure of the original branchial arches, so that there are fissures in the skin leading into fistulæ which open into the pharynx or trachea. The fissure in the skin is small, and is situated about an inch above the sterno-clavicular articulation, usually on one or both sides, more rarely in the middle line. Individual cartilages, as the epiglottis, or one or more rings of the trachea, may be absent, or there may be supernumerary rings. The trachea may divide into three main bronchi instead of two, and in that case two bronchi are given off to the right lung and one to the left. The trachea may be on the left side of the œsophagus, or behind it.

INFLAMMATION.

Acute Catarrhal Laryngitis.—This occurs as an idiopathic inflammation, as a complication of the exanthemata and the infectious diseases, and is produced by the inhalation of irritating vapors and of hot steam and smoke. The inflammation varies in its intensity in different cases. The mucous membrane is at first congested, swollen, and dry; then the mucous glands become more active and an increased quantity of mucus is produced. There is an increase in the desquamation of the superficial epithelial cells, and in the production of the deep cells. A few pus cells are found in the mucus and in the stroma of the mucous membrane. For some reason inflammation of the larynx is frequently attended with spasm of its muscles, thus producing attacks of suffocation. In severe cases, œdema of the glottis may be developed.

After death, the congestion of the mucous membrane frequently disappears altogether.

Chronic Catarrhal Laryngitis.—The surface of the mucous membrane is dry or coated with muco-pus. The epithelium is thickened in some places, thinned in others, or in places entirely destroyed. The stroma is somewhat infiltrated with cells, diffusely thickened, or forming little papillary hypertrophies, or thinned, or necrotic and ulcerated (Fig. 108).

The mucous glands are swollen and prominent. The inflammation may extend to the perichondrium of the cartilages, and thus cause their necrosis. The most severe forms of chronic laryngitis are those associated with pulmonary phthisis.

Croupous Laryngitis occurs most frequently as one of the lesions of diphtheria; it complicates the exanthemata and the infectious diseases. It is produced by the inhalation of irritating gases, hot steam or smoke, and by the introduction of foreign bodies.

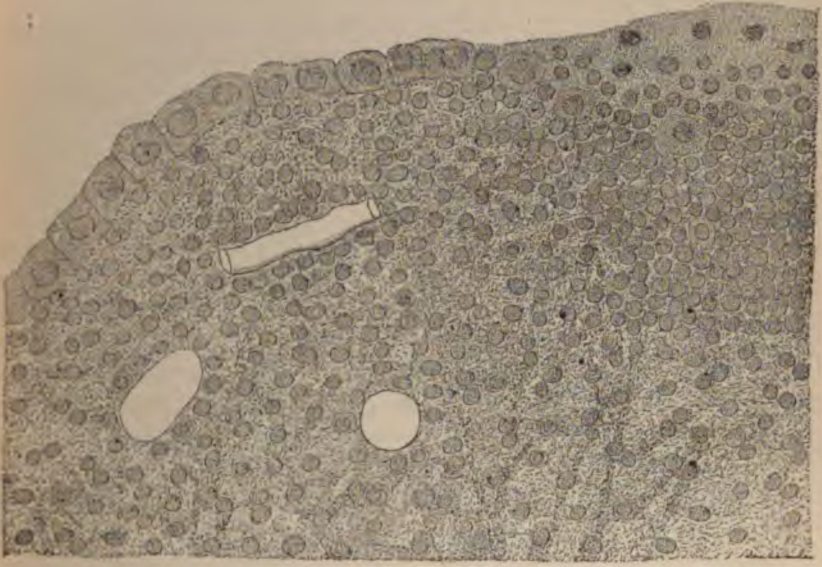

FIG. 108.—AN ULCER OF THE LARYNX IN CHRONIC CATARRHAL LARYNGITIS. × 850 and reduced.

The mucous membrane is swollen and congested. Its surface is coated with fibrin and pus, and its stroma is infiltrated with fibrin and pus. The epithelial cells undergo coagulation necrosis. It is not often that there is necrosis of the deeper tissues.

Syphilitic Laryngitis.—Syphilis often causes laryngitis. The inflammation may have the ordinary characters of an acute or chronic catarrhal inflammation, or it is a productive inflammation with the formation of new tissue in the stroma of the mucous membrane. This new tissue is principally composed of small cells, which often degenerate and die. In this way, the mucous membrane of the larynx and the tissues beneath are thickened in some places and destroyed in others, these changes being especially marked in the upper portion of the larynx. If the perichondrium is involved by these changes, there may be necrosis of the laryngeal cartilages.

Tubercular Laryngitis in its simplest form consists of a catarrhal inflammation, a growth of new cells in the stroma, and the formation of tubercle granula in the stroma without necrosis. The mucous membrane is thickened; it is coated with a layer of mucus, pus, and desquamated epithelium. From the epithelial layer outward, the stroma is infiltrated with cells and with tubercle granula (Fig. 109).

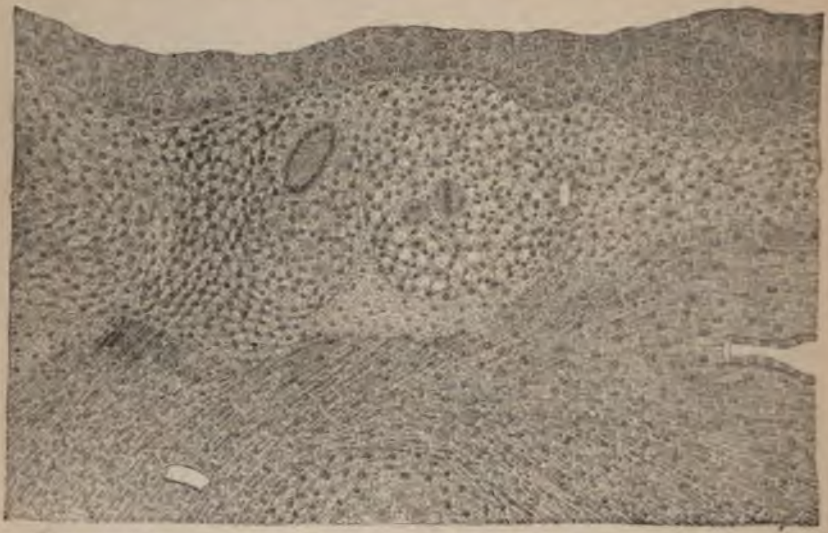

FIG. 109.—TUBERCULAR LARYNGITIS.

When there are added to the production of tubercle tissue an excessive formation of cells and a tendency to necrosis, the conditions become much more serious and complicated. The catarrhal inflammation is intense, with the production of large quantities of pus and mucus. The necrosis results in the formation of ulcers of different sizes and shapes; the inflammation and necrosis extend from the mucous membrane to the wall of the larynx. The epiglottis, the vocal cords, and the adjacent mucous membrane are coated with muco-pus; their surfaces are ragged and irregular. In places the mucous membrane is destroyed, so that ulcers are formed; in places it is thickened and infiltrated with cells and tubercular tissue; in places it is necrotic. In the most severe cases, the entire thickness of the wall of the larynx, with its cartilages, is involved.

Œdema of the Glottis is the name given to serous infiltrations of the mucous membrane of the upper part of the larynx. The swelling is most marked on the posterior wall of the epiglottis, in the aryepiglottidean ligaments and the false vocal cords. In these places, the œdema of the stroma of the mucous membrane may be sufficient to close the larynx.

Acute œdema is due to an inflammatory exudation of serum, and accompanies inflammations of the pharynx, larynx, and neck.

Chronic œdema is of dropsical character and is caused by disease of the heart, pulmonary emphysema, and compression of the veins of the neck.

Acute Suppurative Inflammation may attack the posterior surface of the epiglottis and the aryepiglottidean ligaments. The stroma of the mucous membrane is swollen and infiltrated with serum and pus. Abscesses may be formed in the stroma, which rupture internally, or extend outward into the neck, or into the wall of the pharynx or of the œsophagus. Suppurative inflammation may accompany catarrhal, crupous, tubercular, and syphilitic laryngitis, inflammations and injuries of the pharynx and tonsils; it may complicate typhoid fever and the other infectious diseases.

TUMORS.

Retention cysts of the mucous glands of the larynx may reach such a size as to form sacs projecting into its cavity.

Papilloma is the most frequent form of tumor of the larynx. The tumors grow most frequently from the vocal cords. They consist of a connective-tissue stroma arranged so as to form papillæ covered with epithelium.

Fibromata, lipomata, myxomata, and *angiomata* are occasionally met with.

Chondromata grow from the normal cartilages and are usually multiple and sessile. They may project into the cavity of the larynx.

Sarcomata of the larynx have been seen in a considerable number of cases. They occur both in children and in adults. They are composed of fusiform or round cells, with a stroma which varies in quantity in the different cases.

Carcinomata may invade the larynx from the tongue or the pharynx, or may originate in it. They are composed of flat epithelial cells packed together in the usual way.

In the trachea, tumors are of rare occurrence, but occasional examples of growths similar to those in the larynx have been met with.

THE PLEURA.

HYDROTHORAX.

Non-inflammatory accumulations of clear serum in the pleural cavities are of frequent occurrence. They are produced by the same causes which effect dropsy in other parts of the body—lesions of the heart, liver, and kidneys, and changes in the circulation and in the composition of the blood.

If the amount of serum is small, it is of little consequence; if it is large, it may compress the lower lobes of the lungs and interfere with respiration.

There may be changes in the endothelium of the parietal pleura. Instead of the regular endothelium, large and small flat cells of irregular shape are found.

HÆMORRHAGE.

Extravasations of blood in the substance of the pleura are found in persons who have died after suffering from the infectious diseases; and as the result of injuries to the wall of the thorax.

Blood in large quantity in the pleural cavities is found after rupture of aneurisms of the heart and pericardium.

Bloody serum in the pleural cavities is not often found with ordinary pleurisy. But with tubercular pleurisy and traumatic pleurisy it is not infrequently present.

INFLAMMATION.

The inflammations of the pleura are all spoken of by the common name of pleurisy.

All the different inflammations of the lung are capable of being accompanied by pleurisies, which begin in the pulmonary pleura and extend to the costal.

Besides these, however, there are many pleurisies which belong primarily to the costal pleura and extend from there to the pulmonary pleura.

Such pleurisies occur as idiopathic inflammations, as complications of various diseases, as the result of injuries, or are produced by the inflammation of adjacent parts.

We can distinguish:

I. Pleurisy with the production of fibrin.
II. Pleurisy with the production of fibrin and serum.
III. Pleurisy with the production of fibrin, serum, and pus.
IV. Chronic pleurisy with the formation of adhesions.
V. Tubercular pleurisy.

All the varieties of pleurisy can best be studied in the lesions which are developed in and on the costal pleura. The lesions can be observed in the human subject, and can be produced artificially in the lower animals. It is in these artificial pleurisies especially that we are able to see the early changes produced by the inflammation, and to watch the process step by step.

The free surface of the costal pleura is covered with a single layer of flat cells—the endothelium. The pleura itself is formed of planes of connective tissue, reinforced by elastic fibres. Connective-tissue cells with large bodies and branching processes are present in considerable numbers, being most abundant in the layers beneath the endothelium. In the connective tissue are imbedded blood-vessels, lymphatics, and nerves.

I. Pleurisy with the Production of Fibrin—Dry Pleurisy—Acute Pleurisy.

This form of pleurisy is apt to involve circumscribed areas of the costal, mediastinal, diaphragmatic, or pulmonary pleura, less frequently the entire pleura of one side of the chest. While the inflammation is going on, the affected portion of pleura is coated with fibrin, the surface of the opposite portion of pleura is coated in the same way, and bands of fibrin join the two together. After the inflammation has run its course, we find the affected portion of pleura thickened by the formation of new connective tissue, while bands of connective tissue extend between the opposed pleural surfaces.

As an exceptional condition there is inflammation of the entire pleura of one side, with the production of such an enormous amount of fibrin as to compress the lung and cause death.

II. Pleurisy with the Production of Fibrin and Serum—Pleurisy with Effusion—Subacute Pleurisy.

This is the most common form of pleurisy. As a rule, it involves the greater part of the pleura of one side of the chest. Sometimes, however, the pleura of both sides of the chest is involved, and then the pericardium also is often inflamed.

While the inflammation is in progress, the surface of the affected pleura is coated with fibrin, and bands of fibrin stretch between the parietal and pulmonary pleura. In the pleural cavity is serum in variable quantity. This serum is clear, or turbid from the presence of pus cells and flocculi of fibrin. The lung is compressed in different degrees and positions, according to the quantity of the serum and the character of the adhesions.

If the patients recover, the serum is absorbed, the fibrin disappears, and there are left behind connective-tissue thickenings of the pleura and adhesions.

These two forms of pleurisy, although different in their clinical histories, are yet anatomically essentially the same. In both of them we find a regular sequence of changes. First, the production of fibrin and a few pus cells, either with or without serum. Second, a gradual absorption of the serum and fibrin. Lastly, the formation of permanent new connective tissue in the form of adhesions or of thickenings of the pleura. Throughout the whole process the tissue of the pleura is but little changed; the products of inflammation, although they originate in the tissue of the pleura, do not infiltrate it, but make their way to its surface, there accumulate, and there undergo their different changes. Variations from the regular course of the inflammation are effected by the excessive formation either of the fibrin, the pus, or the serum, and by the manner in which these inflammatory products are absorbed.

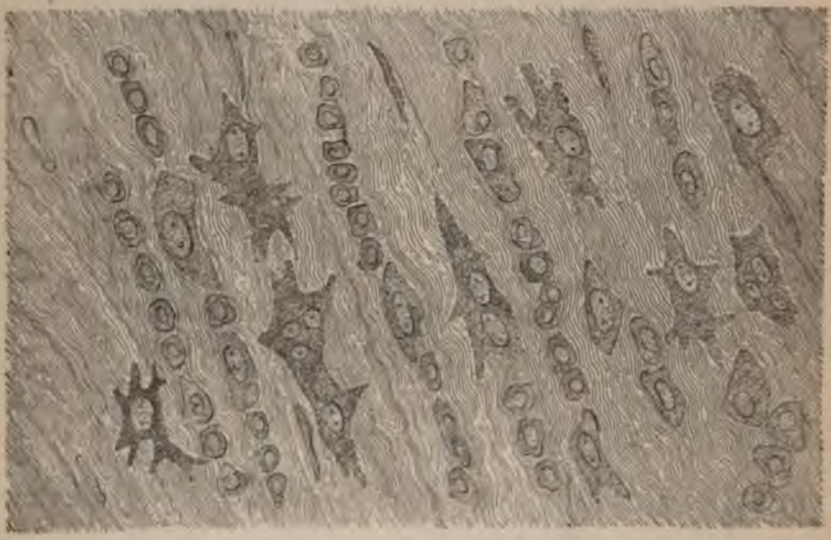

Fig. 110.—An Artificial Pleurisy in the Dog of Twenty-four Hours' Duration, × 750 and reduced.

Swelling and growth of connective-tissue cells in the pleura.

If we endeavor to follow out the successive changes by which the fibrin, pus, and serum make their appearance and then disappear, and the way in which permanent new connective tissue takes their place, we encounter several difficulties. It is impossible to obtain autopsies which will give the lesions belonging to each successive day of the disease; the pleura does not really show well if the patient has been dead more than two or three hours before the autopsy; and in most cases the inflammation is too intense, its products are too abundant, to be easily studied.

To obviate these difficulties we must resort to the lower animals.

By injecting a solution of chloride of zinc into the pleural cavities of dogs, we can excite pleurisies exactly resembling those which we see in the human subject. By varying the amount of fluid injected, we can obtain pleurisies of different degrees of intensity. By using a number of animals, we can observe the course of the inflammation from hour to hour, and from day to day.

In such an artificial pleurisy, the first change is congestion. The pleura is of a uniform bright red color, its surface moist and shining. There is as yet no serum and no fibrin. Already, however, the endothelial cells have fallen off in patches, the superficial connective-tissue cells are swollen and increased in number, and a few pus cells are present. These are all the changes for from half an hour to six hours after the irritant has been applied to the pleura.

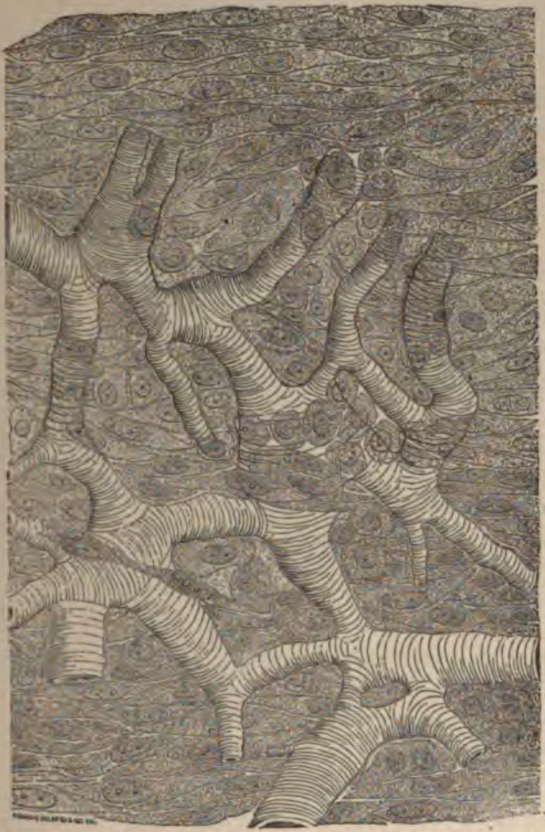

FIG. 111.—ARTIFICIAL PLEURISY IN THE DOG ON THE FIFTH DAY, × 750 and reduced. The layer of new tissue on the surface of the pleura.

The next step in the inflammatory process is the production of serum and fibrin. The serum collects in the bottom of the pleural cavity, the fibrin coats the pleura. As the fibrin is produced the pleura loses its natural moist and shining appearance. The fibrin appears first in the form of little granules, knobs, and threads between the edges of the endothelial cells and overlying them. A few pus cells are entangled in the fibrin and infiltrated in the superficial layers of the pleura. The swelling and new growth of the connective-tissue cells are now well marked. The bodies of the branching cells are swollen, and small polygonal, nucleated cells, arranged in rows between the fibres of the basement substance, make their appearance. By the end of twenty-four hours these changes are fully developed (Fig. 110).

After this the production of fibrin, serum, and new connective-tissue cells continues, and by the third or fourth day the new connective-tissue cells are present, not only in the superficial layers of the pleura, but also in the layer of fibrin coating its surface and forming adhesions.

By the fourth or fifth day, the cells in the fibrin are still more numerous; blood-vessels make their appearance, which can be injected from the arteries of the pleura (Fig. 111).

After this the serum is gradually absorbed. The layer of fibrin and cells on the surface of the pleura exhibits a constant decrease of fibrin and increase of cells, and becomes more intimately connected with the surface of the pleura.

By the fourteenth day the fibrin has disappeared, and a basement substance has been formed between the cells. Of the new cells, the superficial ones are changed into endothelium, the deeper ones into branching cells. The changes in the adhesions between the pulmonary and costal pleura are the same as those in the layer of fibrin coating the costal pleura.

The lesions of human pleurisy seem to be essentially the same as those of the artificial pleurisy just described. But the inflammatory products are formed in larger quantities, a much longer time is required for their absorption, and the formation of new connective tissue follows more slowly.

In these forms of pleurisy, therefore, two distinct processes take place:

1. The blood-vessels are congested, and through their walls transude the plasma of the blood and a few white blood-globules.

2. The superficial connective-tissue cells are increased in size and number.

The products of the first of these processes, the fibrin and serum, are regularly reabsorbed.

The product of the second of these processes, the new connective-tissue cells, regularly increases until a layer of new connective tissue is formed.

The natural termination of such a pleurisy is the recovery of the patient, with thickenings of the pleura and adhesions.

The irregular terminations are: The death of the patient, the protracted existence of the fibrin and serum, and the change of the character of the inflammation so that pus is produced.

III. Pleurisy with the Production of Fibrin, Serum, and Pus—Empyema.

This form of pleurisy may occur under several different conditions.

1. The inflammation is at the very outset of severe character, with the formation of pus.

2. A pleurisy with the production of fibrin and serum, either gradually or suddenly, changes its character, and pus is formed.

3. Phthisical areas of softening, or abscesses of the lung, abscesses in the wall of the thorax, or in the liver, or in the abdomen, rupture into a pleural cavity and set up an empyema.

4. The inflammation may be not only purulent, but also gangrenous in character. The fluid in the pleural cavity, the fibrin and pus coating the pleura, and the pleura itself, may putrefy, with the proliferation of bacteria and the evolution of gases. This may take place either in a closed pleura or in one which has been opened.

5. If there is an opening into a pleural cavity, either through the lung or through the wall of the thorax, there is air in the pleural cavity, in addition to the inflammatory products. Such a condition is called *pyo-pneumothorax.*

In all these different cases, the pleural cavity is partly or completely filled with purulent fluid, and the lung is either compressed against the vertebral column or partly adherent to the chest wall. Sometimes, however, the purulent fluid is shut in by adhesions, either between parts of the lung and the thoracic wall, or between the lung and the diaphragm, or between the lung and the pericardium, or between the lobes of the lung.

The fluid in the pleural cavity is usually a thin, purulent serum, composed of serum, pus globules, endothelial cells, and pieces of fibrin. But sometimes this fluid is very thick and viscid.

In empyema in its earlier stages, the lesions are the same as those in pleurisy with effusion, with the addition of pus in the serum, the fibrin, and the superficial layers of the pleura.

In children, the inflammation may remain in this condition for a long time, but in adults other changes in the pleura are soon developed.

These changes consist in the growth of a large number of small polygonal and round cells, the basement substance is split up, and the pleura is changed into a tissue resembling granulation tissue.

The pleura is thus considerably thickened. Its surface is coated with fibrin and pus, or is bare like the surface of an ulcer.

In this condition the pleura may remain for months or years; its inner layers formed of granulation tissue, its outer layers of dense connective tissue.

Sometimes the cell growth is more active, necrotic changes are added, and so there is a conversion of portions of the pleura into pus. Such a suppuration may extend from the pleura to the fasciæ, the muscles, the skin, the diaphragm, or the lungs. Thus the pus may find an exit, through the wall of the thorax, into the peritoneal cavity or into the lungs.

If the empyema becomes gangrenous, the pleural cavity contains foul gases, the purulent serum is dirty and stinking and swarms with bacteria. The fibrin coating the pleura is of green or brown color. Portions of the pleura itself may also become gangrenous.

In old cases, the thickening of the pleura may reach an enormous degree, and it may become calcified. The perichondrium of the cartilages and the periosteum of the ribs may become inflamed, with necrosis of the cartilages and ribs, or a production of new bone.

Empyema is, therefore, a very much more serious lesion than the two forms of pleurisy just described. The lesions involve not merely the surface of the pleura, but its entire thickness. When the pleura has thus been converted into granulation tissue, it is hardly possible for it to return to a normal condition.

It is important to remember that in children the changes in the pleura itself are less profound, and that in adults they become more and more marked, according to the duration of the disease.

IV. Chronic Pleurisy with the Formation of Adhesions.

This form of pleurisy may follow one of the varieties of pleurisy just described, it may be associated with emphysema and chronic phthisis, or it may occur by itself.

After death the pulmonary and costal pleura are found thickened and joined together by numerous adhesions. These changes may involve only a part or the whole of the pleura on one or both sides of the chest.

The thickened pleura is covered with endothelial cells, which are increased in size and number; the connective-tissue cells in the pleura are also increased in number, and the blood-vessels are more numerous.

The adhesions are formed of connective tissue resembling that of the costal pleura, containing blood-vessels and covered with endothelium.

V. Tubercular Pleurisy.

In acute general tuberculosis, miliary tubercles are often present in the pleura. In acute and chronic phthisis, besides the fibrin, pus, serum, and new connective tissue so often produced, there may also be miliary tubercles, or larger, flat, cheesy nodules.

There are, however, cases of tubercular pleurisy which have the characters of a local tubercular inflammation. Tubercles are either absent altogether from the rest of the body, or of secondary importance to the pleurisy.

This form of pleurisy involves the pleura of one side of the thorax only. It may be rapidly developed, the patient dying at the end of two weeks; or it may continue for months. It seems to be very fatal.

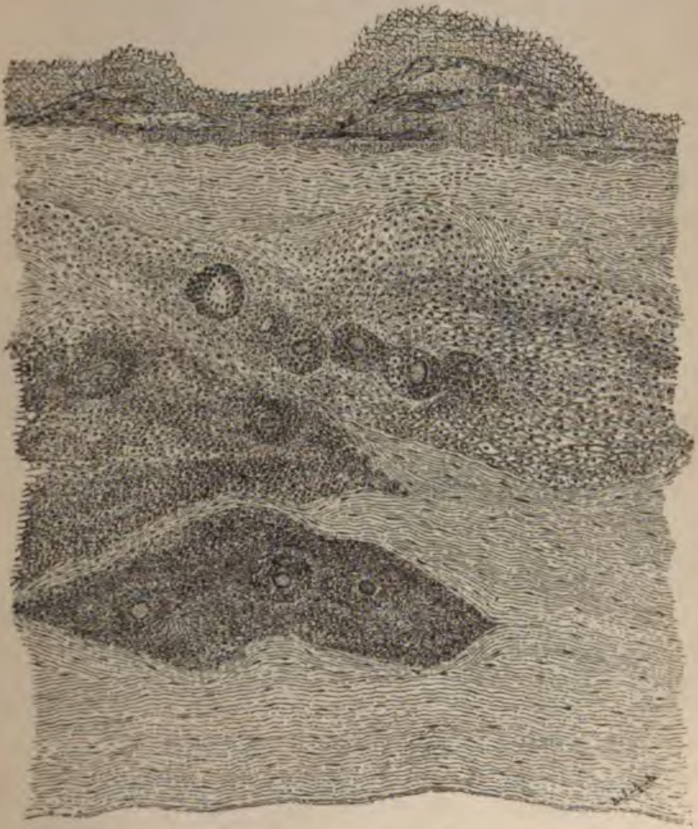

FIG. 112.—TUBERCULAR PLEURISY, × 90 and reduced.
Drawn from a vertical section of the costal pleura.

The inflammation may be confined to the costal pleura, or may involve also the diaphragmatic and pulmonary pleura. The gross appearance of the lesion varies.

1. The pleura is thickened, its surface is bare of fibrin; it is of a bright-red color from the congestion of the blood-vessels, and this red surface is mottled with white dots—the miliary tubercles. In the pleural cavity is bloody serum.

2. The pleura is thickened, it is thickly coated with fibrin, no tubercles are visible to the naked eye; the pleural cavity contains clear serum.

3. The pleura is thickened and the pleural cavity contains purulent serum.

In all the cases, the changes in the pleura itself are essentially the same. The thickened pleura is infiltrated with new connective-tissue cells. Scattered through its entire thickness are tubercle granula, either single or joined together by diffuse tubercle tissue (Fig. 112). The smaller blood-vessels show a growth of their endothelial cells.

NEW GROWTHS.

Fibroma.—Little white or pigmented fibromata of the size of a pin's head are often present in the pulmonary pleura.

Larger fibrous tumors are formed in the deeper layers of the costal pleura, and project into the pleural cavity. They may become detached and are then found loose in the pleural cavity (Lebert).

Lipoma.—Fatty tumors are formed beneath the costal pleura, and project into the pleural cavity (Lebert).

Carcinomata, sarcomata, and *lymphomata* are usually secondary to similar tumors in other parts of the body.

A peculiar form of primary new growth in the pleura has been described by several observers.[1] It is associated with a pleurisy with the production of fibrin and serum. There is a diffuse thickening of the costal pleura, or circumscribed nodules of different sizes.

The new growth seems to begin in the lymphatics of the pleura, which are distended with flat, nucleated cells.

I (Delafield) have seen two of these cases. The first case was a woman, fifty-three years old, who was ill with the symptoms of pleurisy with effusion, for four months. After death, the left pleural cavity was found to be full of bloody, purulent serum. The costal pleura was moderately thickened and coated with a layer of fibrin and pus. Beneath the fibrin and pus was a thin layer of granulation tissue. In this tissue and in the pleura were anastomosing tubules filled with flat, nucleated cells. The tubules looked like lymphatics.

The second case was a man, sixty-three years old, who had the symptoms of pleurisy with effusion, for four months. After death, the right pleural cavity was found half full of bloody serum. The costal, diaphragmatic, and pulmonary pleura were coated with fibrin, and contained numerous white nodules, some of them as large as a pigeon's egg. These nodules were formed of a connective-tissue stroma inclosing irregular spaces and tubules filled with flat, nucleated cells.

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[1] *Birch-Hirschfeld,* "Path. Anat.," p. 768. *E. Wagner,* Arch. d. Heilkunde, xi. *R. Schulz,* Arch. d. Heilkunde, xv. *Thierfelder,* "Atl. d. path. Hist.," 4 Lief.

It is very difficult to class these tumors; whether to call them by the name of Carcinoma, Sarcoma, or Endothelioma is not easy to say.

THE BRONCHI.

INFLAMMATION.

Acute Catarrhal Bronchitis is a disease of very common occurrence, but one which seldom proves fatal. Our knowledge of its lesions is derived from severe cases, from experiments on animals, from cases which are complicated by other diseases, and from the symptoms which we observe during life.

The inflammation involves regularly the trachea and the larger and medium-sized bronchi, less frequently the smaller bronchi also. As a rule, the bronchi in both lungs are equally affected.

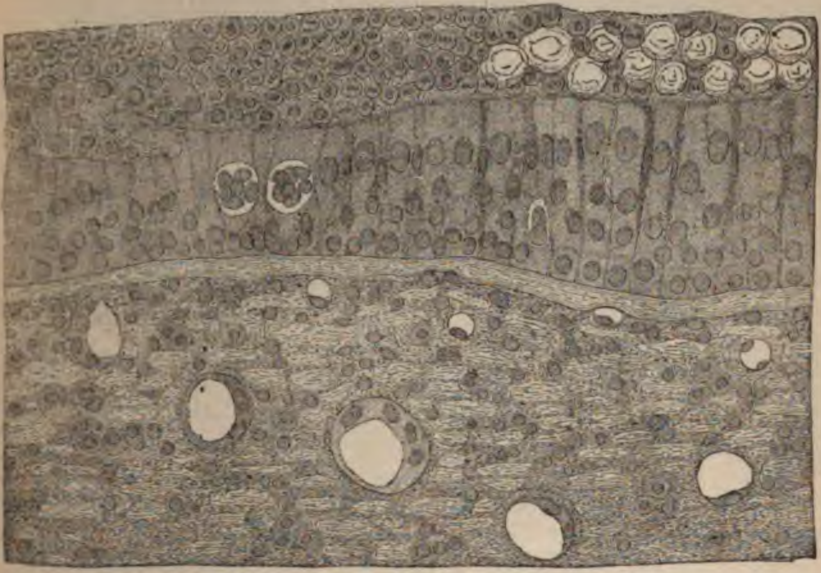

FIG. 113.—ACUTE CATARRHAL BRONCHITIS, × 850 and reduced.

The first change seems to consist in a congestion and swelling of the mucous membrane, with an arrest of the functions of the mucous glands. This is attended with pain over the chest, a feeling of oppression, sometimes spasmodic dyspnœa, and a dry cough. After this, the mucous glands resume their functions with increased activity, the congestion diminishes, there is an increased desquamation of epithelium, an increased formation of the deeper epithelial cells, and a moderate emigration of white blood-cells. Sometimes the red blood-cells also escape from the vessels. The patient now has less pain and oppression, the cough is accompanied with an expectoration of mucus mixed with epithelium, pus, and sometimes blood.

After death, the only lesions visible are the increased amount of mucus, the growth of new epithelium, mucous degeneration of the epithelial cells, a few pus cells infiltrating the stroma, and the general congestion of the mucous membrane. The whole process is a superficial one, not producing any changes in the walls of the bronchi beneath the mucous membrane (Fig. 113).

When the inflammation involves the smaller bronchi also, they may be full of pus, but their walls are unchanged.

The filling of the small bronchi may result in the collapse of the groups of air vesicles to which they lead, and thus are produced areas of atelectasis, which may be further changed by inflammatory processes.

Chronic Catarrhal Bronchitis.—This form of bronchitis may be the sequel of one or more attacks of acute bronchitis. More frequently it is associated with emphysema, heart disease, interstitial pneumonia, phthisis, pleuritic adhesions, or the inhalation of irritating substances.

There is, in most cases, a constant production of mucus, pus, and serum in considerable quantities, and these inflammatory products may have a very foul odor. Less frequently these products are very scanty—dry catarrh.

In examining the bronchi in these cases after death, we are often struck by the want of proportion between the symptoms and the lesions. The same bronchi which during life were constantly producing large quantities of inflammatory products and injuring the patient's health, after death may be but little changed from the normal. In other cases, however, the lesions are more marked.

The bronchi contain mucus and pus; they may be congested; their walls are often trabeculated. The epithelium is deformed and desquamating, with a production of new cells in the deeper layers. The mucous glands are enlarged or atrophied. The connective-tissue stroma is thickened and infiltrated with cells. The coats of the arteries in the walls of the bronchi may be thickened. There may be cylindrical dilatation of one or more bronchi. The muscular coat may be thickened or thinned. Very frequently the epithelial cells of the air vesicles and air passages are increased in size and number.

Acute Croupous Bronchitis occurs as a lesion of diphtheria, as associated with croupous laryngitis, as the result of the inhalation of hot steam, with lobar pneumonia, and sometimes as an idiopathic disease.

The bronchi are lined or filled with a mass of fibrin, pus, and desquamated epithelium. Fibrin and pus may also be found beneath the epithelium and infiltrated in the stroma.

Chronic Croupous Bronchitis is attended with the formation in one or more bronchi of masses of fibrin which are expectorated by the patient in the form of branching casts of the bronchi. The disease is a very chronic one, and is often associated with phthisis. After death, the bronchi are said to be found but little altered from the normal.

Curschmann[1] has described under the name of "bronchiolitis exudativa" a form of bronchitis in which small threads and bands of gray or yellow, partly transparent, coagulated matter are formed in the small bronchi. Vierordt[2] has found similar formations in lobar pneumonia. Leyden and Levi have found them in broncho-pneumonia.

In different forms of bronchitis, especially in those associated with asthma, the exudation may contain small, octahedral bodies, probably composed of mucin. They are accidental formations, probably formed from cells, and may be found in the sputa.

BRONCHIECTASIA.

Dilatation of the bronchi presents itself under three forms: the cylindrical, the fusiform, and the sacculated.

The cylindrical dilatation is a uniform enlargement of one or more bronchi for a considerable part of their lengths. It is found in bronchi of every size, but most frequently in the medium sized.

The fusiform dilatation is a mere variety of the cylindrical. The bronchus is uniformly dilated for a short distance, and then resumes its natural size. Several such dilatations may be found in the same bronchus.

The sacculated dilatations form the largest cavities. These cavities communicate with one side of the bronchus; the peripheral portion of the bronchus may be obliterated. The bronchus leading to the cavity may be of normal size, or dilated, or stenosed, or even completely obliterated. Such sacculated dilatations may reach a very large size, and may communicate with each other.

Any inflammatory process which involves the thickness of the wall of a bronchus seems to be capable of producing dilatation of that bronchus.

In acute general bronchitis and broncho-pneumonia in children, cylindrical dilatation of a number of the medium-sized bronchi is often produced.

In the persistent broncho-pneumonia of children, such dilatations reach a still greater development.

In acute and chronic phthisis, tubercular inflammation gives rise to sacculated dilatations, which expand with time and are made still larger by the destruction of the adjacent lung tissue.

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[1] Deutsch. Arch. f. klin. Med., xxxii.

[2] Berl. klin. Wochensch., 1883. *B. Levi*, Zeitsch. f. klin. Med., ix. *Leyde* Virch. Arch., Bd. 74.

Chronic bronchitis may lead to cylindrical or sacculated dilatations, sometimes of great size.

Occlusion of some of the bronchi, consolidation of portions of the lung, and extensive pleuritic adhesions, may also produce bronchiectasia.

The walls of these dilatations may preserve the characters of the wall of the bronchus, more or less altered by inflammation (Fig. 114), or these characters may be altogether lost. The dilatations may contain mucus and pus, or they may be empty.

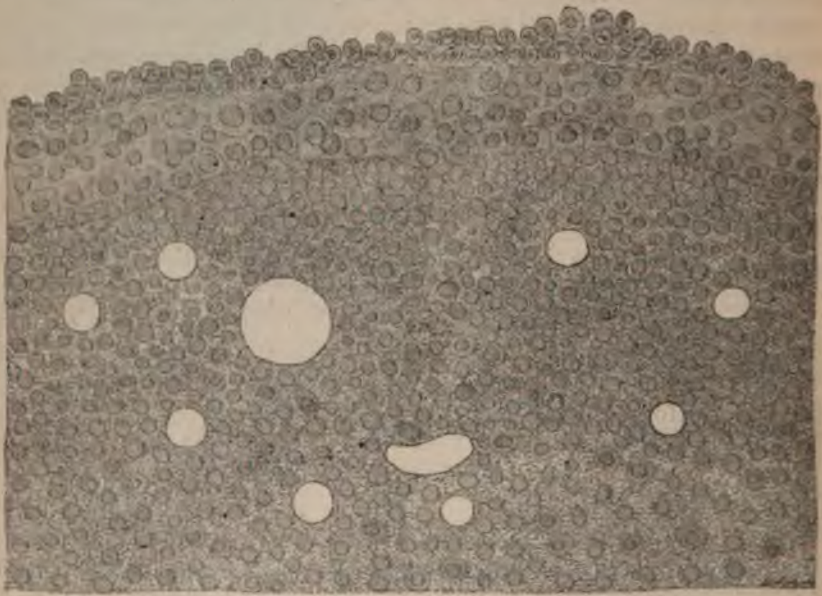

FIG. 114.—SECTION OF THE WALL OF A BRONCHIECTASIA, × 850 and reduced.

NEW GROWTHS.

Ossification of the walls of the bronchi is sometimes found.

Lipoma in the submucous connective tissue has been described by Rokitansky.

Carcinoma of the walls of the bronchi may occur as a secondary lesion, but as a primary growth it is very rare. It may be formed in the large or small bronchi, follow the course of the bronchial tree, or extend to the lung tissue.

Langhans[1] describes a primary carcinoma of the lower end of the trachea and the large bronchi in a man forty years old. The lower end of the trachea and the large bronchi showed a general thickening of their walls, with flat tumors projecting inward. The new growth was composed of a stroma inclosing cavities filled with cells. The cells were small, nucleated, polygonal or cylindrical in shape, and packed closely together. Apparently the new growth originated in the mucous glands.

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[1] Virchow's Arch., liii., p. 470. Virchow's Arch., Bd. 56, Bd. 85. Arch. d. Heilkunde, xix. Prager med. Wochensch., 1883. Zeitsch. f. Heilkunde, v.

I (Delafield) have seen one case of new growth of the lower end of the trachea and large bronchi which I hardly know how to name. The patient was a woman, twenty-seven years old, who suffered for seven months from cough, constantly increasing dyspnœa, and progressive emaciation. After death, the wall of the lower end of the trachea was found to be thickened. Its inner surface was studded with minute papillary outgrowths. The epithelial cells were increased in size and number, and in some places were replaced for considerable spaces by a regular layer of pavement epithelium. The mucous glands were hypertrophied. The wall of the treachea from the basement membrane to the cartilages was thickened by new connective tissue.

The walls of the large bronchi were very much thickened and their cavities nearly obliterated. The entire wall was thickened with new connective tissue. The epithelial layer was thickened, and in places replaced by patches of pavement epithelium. The mucous glands were enlarged.

Sarcomata of the walls of the bronchi occur as secondary growths, and as prolongations of similar growths in the mediastinum.

Primary sarcoma of the bronchi seems to be rare.

Hesse[1] describes a form of lympho-sarcoma, forming nodules around the bronchi, as of common occurrence among the miners in some cobalt and nickel mines.

THE LUNGS.

MALFORMATIONS.

One or both lungs may be entirely wanting, or only partially developed. In some of the cases with only one lung, the patients have grown up to adult life.

A peculiar degeneration, by which the lung is converted into a number of sacs containing air and serum, the sacs communicating with the bronchi, has been seen in a few instances.

The lobes may be subdivided by deep fissures. An accessary lobe, separated from the lung, between the base of the left lung and the diaphragm, has been described by Rokitansky.

There may be hernia of the lung, with absence of part of the wall of the thorax.

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[1] Arch. f. Heilkunde, xix., p. 160.

There may be transposition of the lungs, with similar changes in the position of the heart and the abdominal viscera.

INJURIES—PERFORATIONS.

Severe contusions of the thorax may produce rupture of the lungs, with extravasation of blood into the pleural cavities.

The lungs may be wounded by a fractured rib and by penetrating weapons and projectiles. Such injuries often produce bleeding into the lung tissue and inflammatory changes. The lungs, however, exhibit a considerable degree of tolerance for such injuries, and the patients often recover.

Collections of pus in the pleural cavities, the mediastinum, the liver, the spleen, the kidneys, and the peritoneal cavity may perforate the lungs.

CONGESTION AND ŒDEMA.

These two conditions are regularly associated with each other in the lungs, although one or the other of them may preponderate in different cases.

A moderate degree of congestion and œdema of the posterior portions of the lungs is often found as a result of post-mortem changes.

In persons who have been comatose from any cause for some hours before death, congestion and œdema of the lungs are regularly developed.

With disease of the heart, kidneys, and lungs, the congestion, and especially the œdema, may be excessive. The lungs may be so completely infiltrated with serum as to be unaërated. Such a solid œdema of the lungs is sufficient of itself to cause death. It has been asserted by Welch[1] that the cause of such an excessive œdema is a paralysis of the left side of the heart, while the force of the right heart is unimpaired. Such an explanation seems to be plausible.

Patients confined to bed for a considerable length of time may develop congestion of the dependent portions of the lungs—*hypostatic congestion.* The affected portion of lung is shrunken, congested, and imperfectly aërated.

HÆMORRHAGE.

Extravasations of blood within the air cavities are found with the general diseases which produce a disposition to bleeding in different parts of the body.

Blood from the bronchi or from cavities may be inspired into the air vesicles.

Valvular lesions of the heart, especially of the mitral valve, are often accompanied by the production of hæmorrhagic infarctions in the lungs.

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[1] Virchow's Archiv, 72.

These infarctions are circumscribed, of rounded or wedge-shaped form. from the size of a walnut to that of an orange. They are of dark-red color, unaërated. the air passages distended with blood, and are often surrounded by a zone of pneumonia They may be situated in any part of the lungs, but are most common in the lower lobes. When they are near the surface of the lungs, a circumscribed pleurisy is often produced.

Such infarctions may produce death; they may become gangrenous, or the blood may become absorbed, or they may be gradually changed into a smaller mass of pigmented fibrous tissue.

It is probable that these infarctions are produced either by thrombosis of branches of the pulmonary artery, or by rupture of the capillaries.

Infarctions of smaller size, and with more disposition to be surrounded by inflammatory changes, are produced by emboli from the right side of the heart, and from thrombi in the veins of pyæmic patients. These infarctions are usually situated near the surface of the lung.

Hæmorrhages with rupture of the lung tissue are produced by severe contusions, by penetrating wounds, and by the rupture of aneurisms.

EMPHYSEMA.

Emphysema is of two kinds—interlobular and vesicular.

Interlobular Emphysema is produced by the rupture of air spaces and the escape of air into the interstitial tissue of the lung. Or the pulmonary pleura may also be ruptured and the air escapes into the pleural cavity, or into the mediastinum and from thence into the neck. Such a rupture of the air spaces is most frequently caused by broncho-pneumonia with consolidation of portions of the lungs.

Vesicular Emphysema is a dilatation of the air passages and vesicles of the lungs. A temporary emphysema can be produced in a variety of ways. The bronchi may be obstructed in such a way that the air can enter the air spaces, but cannot escape from them. A portion of the lungs may be consolidated or compressed, and then the air spaces of the rest of the lungs will be dilated. Death may take place with a dilatation of the lungs, which remains after death.

Permanent emphysema may change an entire lung if the other lung becomes permanently unaërated; it may change portions of a lung if other portions are consolidated.

"Substantive emphysema" is a term which is now used in a clinical rather than in an anatomical sense. It is used to designate a group of cases in which there are regularly developed changes in the shape of the thorax, certain characteristic physical signs, a liability to bronchitis, to constant and spasmodic dyspnœa, to venous congestion of the viscera and of the skin. In patients who present such symptoms during life, we find after death diffuse changes of both lungs, of which dilatation of the air spaces may form a part. If the dilatation of the air spaces does exist, the term "substantive emphysema" is appropriate; if it does not exist, we employ a term which contradicts itself.

The real lesion of substantive emphysema is a chronic productive inflammation of the lung with the formation of new connective tissue—a process analogous to similar chronic inflammations of the endocardium, arteries, and kidneys, and one which, like them, may constitute a formidable disease or an unimportant senile change.

Both lungs are moderately or considerably increased in size. Very often they are partly covered by connective-tissue pleuritic adhesions. The mucous membrane of the bronchi may be coated with mucus or with muco-pus. The muscular coat of the bronchi may be thickened; their entire wall may be thickened and infiltrated with cells; they may be narrowed or dilated; they may be surrounded by zones of pneumonia. The cells which line the walls of the air spaces are increased in size and number. The walls of the air spaces are more or less thickened, except in the case of some of the air spaces which are dilated. In the walls of some of the air spaces, those which are thickened as well as those which are thinned, are formed small holes (Fig. 115) which may later reach a large size, so that adjacent air spaces become fused together.

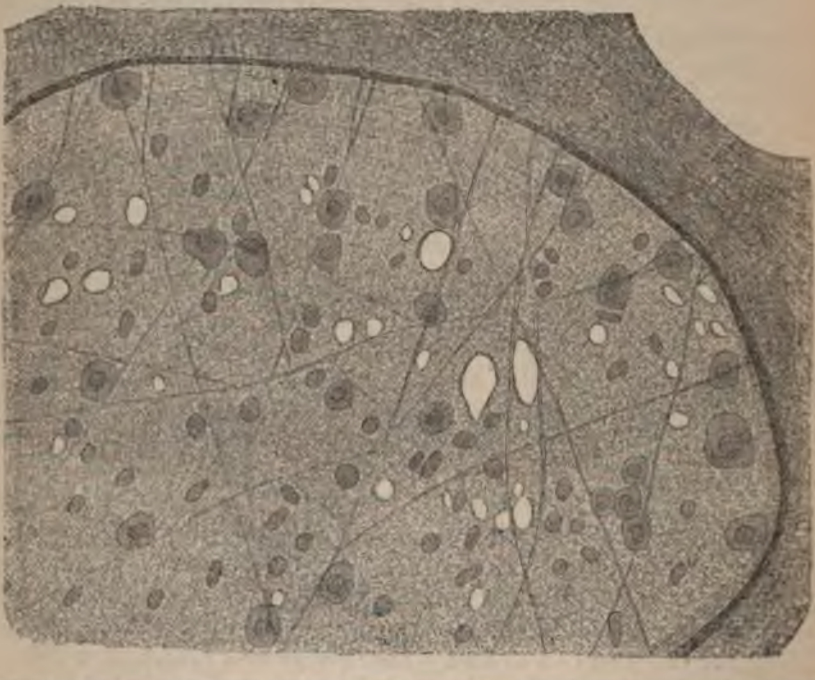

FIG. 115.—EMPHYSEMA, SHOWING HOLES IN THE WALLS OF THE AIR VESICLES, × 850 and reduced. From a case of chronic miliary tuberculosis.

In some cases of substantive emphysema, no dilatation of the air spaces exists. In many of the fatal cases, the dilatation is but moderate; in some cases it is very marked. The dilatation may involve the air passages alone, or both the air passages and the vesicles. It is not uniform, but involves some parts of the lungs more than others.

The arteries throughout the lungs and in the walls of the larger bronchi may have their coats thickened. The capillaries in the walls of the air spaces which are but little dilated are unchanged. Those of the dilated air spaces are separated by wider intervals; they may be smaller; it is said that they may be obliterated.

The right ventricle of the heart may be dilated or hypertrophied, or both. There may be venous congestion of the pia mater, the stomach, the small intestine, the liver, the spleen, the kidneys, and the skin. There may be dropsy.

ATELECTASIS.

A collapsed and unaërated condition of portions of lung tissue is either congenital or acquired.

(1) In congenital atelectasis, portions of the lung are firm, non-crepitant, of a dark blue or purple color, depressed and smooth on section. These portions can usually be artificially inflated, and then cannot be distinguished from the surrounding pulmonary tissue. This condition is produced by the inability of the child after birth to fully inflate its lungs, either from want of sufficient vitality or from obstruction of the bronchi. If the child lives for some time, and the collapsed lobules are not inflated, they become hard and dense.

(2) In young children, the smaller bronchi may become obstructed by the inflammatory products of bronchitis, and the corresponding air vesicles will then collapse. We then find scattered through the lungs collapsed lobules like those in the new-born child. Inflammatory changes may be subsequently developed in the collapsed lobules.

(3) In adults, large or small portions of lung tissue may become collapsed as the result of bronchitis, of stenosis of a large bronchus, of compression of a bronchus, of paralysis of the pneumogastric, of compression of the lungs by fluid or by new growths, and of long-continued feebleness of the act of respiration.

GANGRENE OF THE LUNGS.

It is customary to distinguish two forms of gangrene of the lung: the circumscribed and the diffuse; yet both can occur together.

Circumscribed gangrene occurs in the form of one or more rounded or irregular masses of variable size. The gangrenous portion of lung is at first brown and dry. The surrounding lung tissue is congested or œdematous, or infiltrated with blood, or inflamed. If the gangrenous focus is near the pleura, the latter will be coated with fibrin. Gradually the gangrenous portion of lung assumes a dirty green color and a putrid odor. It becomes soft, broken down, and separated from the surrounding lung. The blood-vessels may be obliterated by thrombi, or eroded, so that there are profuse hæmorrhages.

Such a gangrenous process may extend to the adjacent lung tissue, or a zone of gray or red hepatization or of connective tissue may be formed.

The fluid from the gangrenous lung may pass into the bronchi and be expectorated; or it may run from one bronchus into another, and set up new gangrenous foci or diffuse gangrene.

The pulmonary pleura may be perforated, and a gangrenous pleurisy produced.

Diffuse gangrene may follow the circumscribed form; it may complicate lobar pneumonia, or occur as an idiopathic condition. A large part of a lobe or of an entire lung becomes greenish, putrid, and soft, and the pulmonary pleura is inflamed. There may be hæmorrhages from eroded vessels. There may be general septicæmia.

Various forms of bacteria may be present in gangrenous areas of the lungs. Among those frequently present is the Staphylococcus pyogenes aureus.

PNEUMONIA.

The inflammations of the lung, as distinguished from those of the bronchi and pleura, are called "pneumonia."

In the present state of our knowledge, the classification of the different forms of pneumonia must be an arbitrary one. We describe separately:

Acute lobar pneumonia.
Broncho-pneumonia.
Secondary and complicating pneumonia.
The pneumonia of heart disease.
Interstitial pneumonia.
Tubercular pneumonia.
Syphilitic pneumonia.

I. Acute Lobar Pneumonia.

This is an acute exudative inflammation, which involves regularly the whole of one lobe, or the larger part of one lung, or portions of both lungs. It is an infectious inflammation, attended with the growth in the lung of pathogenic bacteria; most frequently the "Diplococcus pneumoniæ" of Fraenkel, less frequently the bacillus described by Friedländer. Apparently there are also irregular forms of lobar pneumonia attended with the growth of other species of bacteria.

The inflammation is of pure exudative type, characterized by congestion, emigration of white blood-cells, diapedesis of red blood-cells, and exudation of blood-plasma, while the tissue of the lung itself is but little changed.

During the first hours of the inflammation, only irregular portions of the lobe which is to be inflamed are involved; later, the entire lobe. The lung is congested, œdematous, tough, but not consolidated. The air spaces contain granular matter, fibrin, pus-cells, red blood-cells, and epithelial cells. The epithelium remaining on the walls of the air spaces is swollen; there are large numbers of white blood-cells in the capillaries. The larger bronchi are congested, dry, or coated with mucus; the small bronchi contain the same inflammatory products as do the air spaces. The pulmonary pleura, as a rule, is not coated with fibrin. This is called the stage of "congestion." The stage of congestion regularly only lasts a few hours, but it may be protracted for several days.

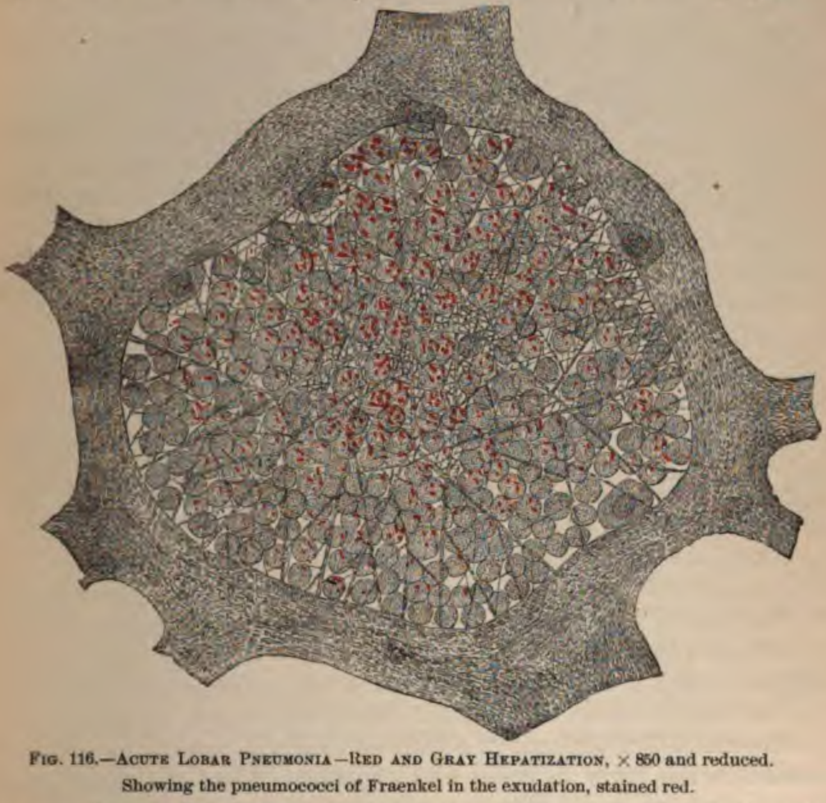

FIG. 116.—ACUTE LOBAR PNEUMONIA—RED AND GRAY HEPATIZATION, × 850 and reduced. Showing the pneumococci of Fraenkel in the exudation, stained red.

When the exudation of the inflammatory products has reached its full development, the presence of these products within the air spaces and bronchi causes the lung to be solid, and at this time the lung is said to be in the condition of "red hepatization." The lung is now consolidated, red, its cut section looks granular, the granules corresponding to the plugs of inflammatory matter within the air spaces. For some time after death the inflammatory products remain solid and the cut section of the lung dry; but later, with the commencement of post-mortem changes, these products soften and the cut section is covered with a grumous fluid. The air vesicles, the air passages, the small bronchi, and sometimes the large bronchi, are filled and distended with fibrin, pus-cells, red blood-cells, and epithelium, and may contain large numbers of bacteria (Fig. 116). In spite of the pressure on the walls of the air spaces, the blood-vessels in their walls remain pervious. The pulmonary pleura is coated with fibrin and the interstitial connective tissue of the lung is infiltrated with fibrin. The hepatized lobe is increased in size, sometimes so much so as to compress the rest of the lung. About one-fourth of the fatal cases die in the stage of red hepatization, at any time from twenty-four hours to eleven days after the initial chill.

After the air spaces have become completely filled with the exudation, if the patient continues to live, there follows a period during which the exudate becomes first decolorized and then degenerated. This is the period of "gray hepatization." The lung remains solid, its color changes, first to a mottled red and gray, then to a uniform gray. The coloring matter is discharged from the red blood-cells and the exudate begins to degenerate and soften. The lung is found passing from red to gray hepatization at any time between the second and the eighteenth day of the disease. It is found completely gray at any time from the fourth to the twenty-fifth day. About one-half of the cases die in the condition of mottled red and gray hepatization; about one-fourth in the condition of gray hepatization.

If the patients recover, the exudate undergoes still further degeneration and softening and is removed by the lymphatics. This is the stage of "resolution." It should commence immediately after defervescence and be completed within a few days. But it may not begin until a number of days after defervescence, or it may be unusually protracted.

The pneumococcus of Fraenkel (often also called the Diplococcus pneumoniæ of Fraenkel and Weichselbaum) is the bacterium most often present in the lungs in acute lobar pneumonia, and the form which there is much reason to believe, in the large proportion of cases, to be the cause of the disease. This germ is, in general, round or oval in shape, and is apt to occur in pairs or often in longer and shorter chains (see Fig. 116). In the tissues, the individual bacteria are frequently smaller at one end than at the other, so that they appear lanceolate. They are, in the tissues, surrounded by a gelatinous capsule. The Diplococcus pneumoniæ is readily stained by Gram's method.

It may be artificially cultivated, but its growth is very meagre below 35°–37° C., or the temperature of the body. As at this temperature gelatin is fluidified, it is usually grown on agar. On this it forms a delicate grayish, transparent growth which is very sensitive to the reaction of the nutriment, requiring a very slight alkalinity. The growth, at best, is not voluminous. It does not apparently grow at all on potatoes. In beef tea it forms a uniform cloudiness and a granular sediment.

A very marked peculiarity of this germ is that it rapidly loses its virulence in artificial cultures. Inoculated subcutaneously into rabbits, mice, and guinea-pigs, the pure cultures usually induce a fatal septicæmia but no pneumonia. If, however, the virulence of the organism be reduced by artificial cultivation, the subcutaneous inoculation may induce a typical pneumonia, as may also the intrapulmonal injection of the virulent cultures.

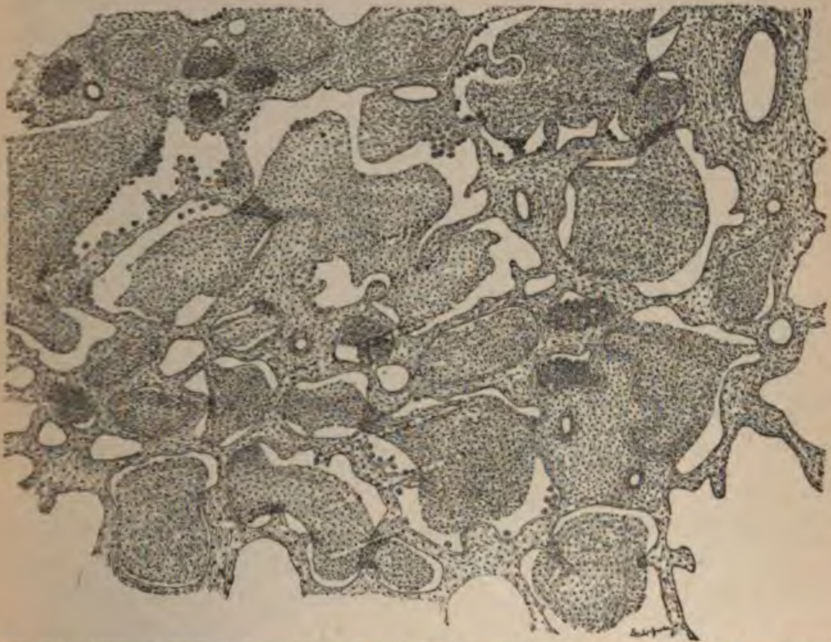

FIG. 117.—ACUTE LOBAR PNEUMONIA WITH THE PRODUCTION OF ORGANIZED TISSUE IN THE AIR SPACES, × 130 and reduced.

The section shows a number of air vesicles containing organized tissue.

An organism identical with the Diplococcus pneumoniæ appears not infrequently to occur in the mouths of healthy persons.[1]

There is a form of lobar pneumonia in which the inflammation is not simply an exudative one, but there is also a growth of new connective tissue in the walls of the air spaces and in their cavities (Fig. 117).

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[1] For a résumé of the studies on the bacterial origin of acute lobar pneumonia, consult *Baumgarten's* "Mykologie," page 236 *et seq.*

This condition has been usually described as a chronic inflammation following an ordinary lobar pneumonia. It seems really to be from the outset a special form of pneumonia. For we find, in patients who have not been sick for more than a few days, that the pneumonia already has its characteristic form. Still further, even in its earlier stages the clinical history is somewhat different from that of an ordinary lobar pneumonia.

If the patient dies within three weeks of the commencement of the pneumonia, we find one or more lobes consolidated but not much enlarged. The hepatization is smooth and dense. The walls of the air spaces are thickened and coated with an increased number of epithelial cells. Some of the air spaces contain only fibrin and pus, but in others there is new connective tissue, basement substance and cells (Fig. 118). In this new tissue there may be new blood-vessels, which can be artificially injected from the vessels of the lung (Fig. 119).

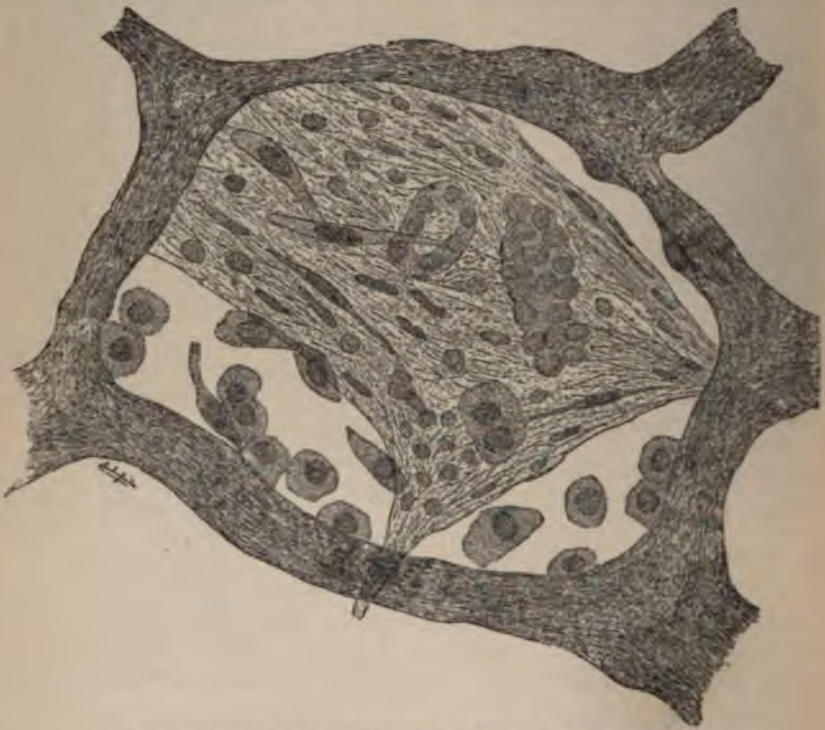

FIG. 118.—ORGANIZED TISSUE IN AN AIR VESICLE, × 850 and reduced.

If the patient lives for several months, we find the lung very dense and smooth. The growth of new connective tissue is more extensive, the air spaces are completely filled, their walls are much thickened, and in some places the lung tissue is completely changed into smooth connective tissue.

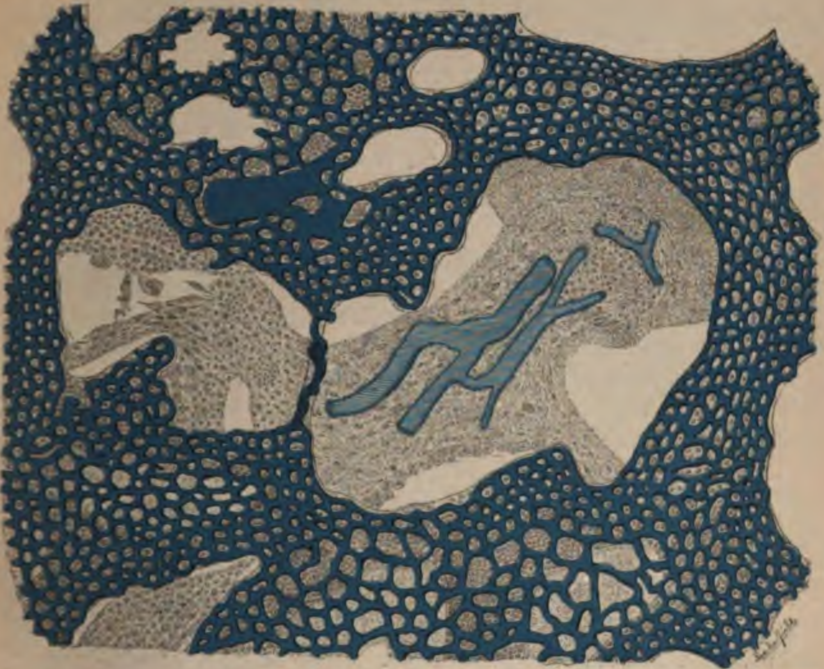

FIG. 119.—AIR VESICLES CONTAINING ORGANIZED TISSUE IN LOBAR PNEUMONIA, × 350 and reduced. The blood-vessels are injected with blue.

II. Broncho-pneumonia (Capillary Bronchitis, Lobular Pneumonia, Catarrhal Pneumonia).

This is the ordinary pneumonia of young children; it is frequent also in young persons, but not as common in adults.

In children, it seems to be due to the same causes which produce lobar pneumonia in adults: to the poisons of the different infectious diseases; to the inhalation of irritating substances and gases, and of the bacteria which produce suppuration.

In adults, the disease may present itself to us in a variety of ways:

1. The patients have an ordinary attack of catarrhal bronchitis lasting for several days. Instead of getting well promptly, however, the patients continue to cough and to feel sick, and, on examining the chest, we find a circumscribed area where there is dulness on percussion and loud, high-pitched voice. This consolidation of the lung does not, however, last very long, and the patients make a good recovery.

2. The patients are suddenly attacked with a very severe and general broncho-pneumonia. There are chills, a rapid rise of temperature,

headache, pains in the back and chest, vomiting, great prostration, a rapid pulse which soon becomes feeble, very bad breathing—rapid, labored, and insufficient—venous congestion of the skin and of the viscera, cough, at first dry, then with profuse mucus and blood-stained sputa, sleeplessness, restlessness and delirium, and albumin in the urine. There are coarse subcrepitant and crepitant râles over both lungs, sibilant and sonorous breathing; the percussion note is normal, or exaggerated, or dull. The disease lasts for from seven to fourteen days, and is very apt to prove fatal.

3. There is a form of broncho-pneumonia in adults which resembles lobar pneumonia. There is a general catarrhal bronchitis, with broncho-pneumonia and consolidation of one or more lobes. The symptoms and physical signs are like those of lobar pneumonia, but with some difference. The invasion of the disease is not as sudden, the pulse is more rapid, the cerebral symptoms are more constant, the expectoration is like that of bronchitis, the physical signs are more slowly developed, the duration of the disease is rather longer and resolution is slower.

4. There is a form of broncho-pneumonia which resembles acute phthisis. The patients have a cough with expectoration, at first mucous, afterward muco-purulent. There is a moderate fever, with evening exacerbations and sweating at night. The patients steadily lose flesh and strength. The physical signs are those of bronchitis and of consolidation of parts of the lung. The disease is protracted, continuing as long as ten weeks, and is apt to prove fatal.

With substantive emphysema there may be developed a subacute or chronic broncho-pneumonia.

The essential or constant lesion of broncho-pneumonia is an inflammation of the walls (not the mucous membrane) of the bronchi, and of the air spaces immediately surrounding the inflamed bronchi. The walls of the bronchi are thickened and infiltrated by a growth of new cells. The walls of the air spaces are thickened, their cavities are filled with fibrin, pus, and epithelium, or with new connective tissue. The inflammation involves the medium sized and smaller bronchi of both lungs, but is not everywhere equally severe; in some parts of the lungs the lesions are much more marked than in others. In some of the cases there are no other changes, except some general congestion of the lungs. In other cases there may be added a catarrhal inflammation of the mucous membrane of the bronchi, diffuse consolidation of parts of the lung, pleurisy, dilatation of the inflamed bronchi, areas of atelectasis, simple or tubercular inflammation of the bronchial glands.

The trachea and the larger bronchi are congested and coated with mucus. The smaller bronchi contain pus, their walls are thickened and infiltrated with cells, and they may be dilated. Around many of the small bronchi are narrow zones of congestion or hepatization. The rest of the lungs is congested and œdematous.

Or the zones of peribronchitic pneumonia are larger, so that a section of the lung is mottled with little whitish nodules, each nodule corresponding to a cut bronchus surrounded by its zone of pneumonia.

Or between these zones of peribronchitic pneumonia are areas of diffuse hepatization which render portions of the lung completely solid (Fig. 120).

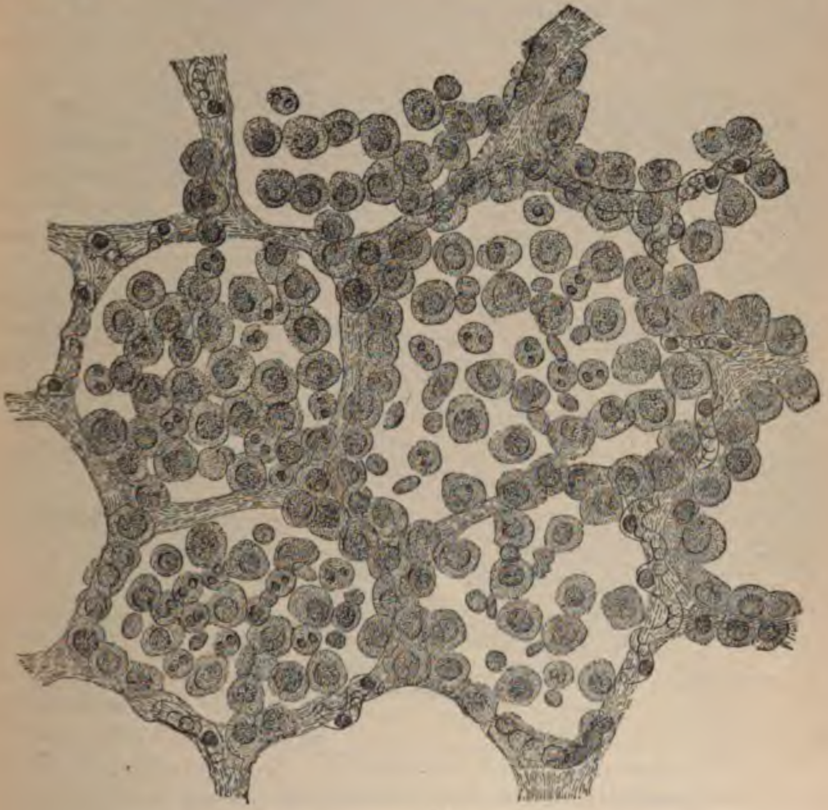

FIG. 120.—BRONCHO-PNEUMONIA IN A CHILD, × 750 and reduced.
Air vesicles in diffuse hepatization.

Or there may be areas of atelectasis corresponding to occluded bronchi.

There is often a thin layer of fibrin on the pulmonary pleura. The bronchial glands are the seat of simple or tubercular inflammation.

The dilatation of the bronchi is not constant. When present, it is of the cylindrical character and involves the medium-sized bronchi for a considerable part of their length. Such dilated bronchi are each of them surrounded by a narrow zone of pneumonia; the intervening lung tissue may be still aërated or hepatized.

In these peribronchitic zones of pneumonia, the thickening and cellular infiltration which exist in the walls of the bronchi extend also to the walls of the air spaces. These walls are thickened and infiltrated with cells, while the cavities of the vesicles are filled with pus and epithelium, or with tissue resembling granulation tissue (Fig. 121). In the diffuse hepatization, the air vesicles are filled with epithelium, pus, and fibrin in varying proportion and quantity; the walls of the air spaces remain unchanged.

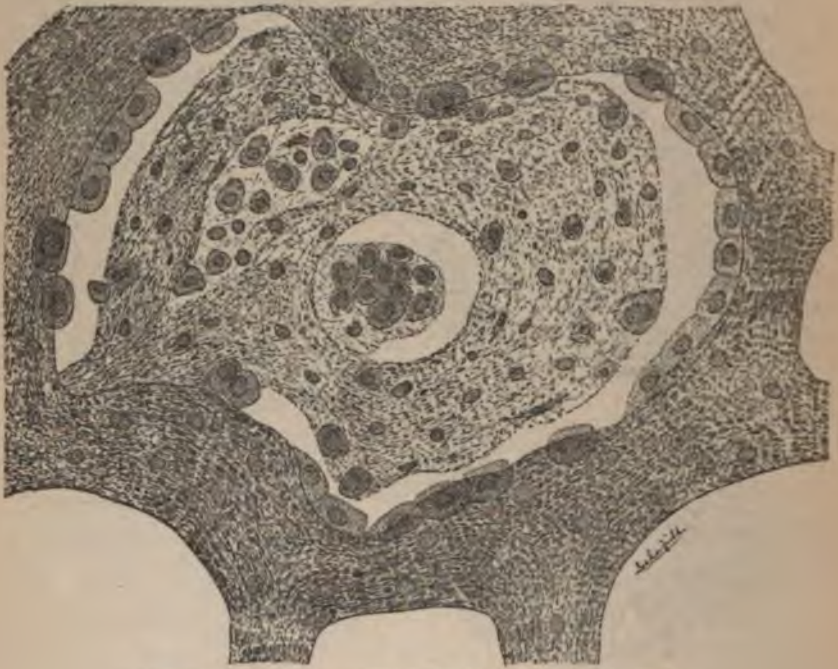

FIG. 121.—BRONCHO-PNEUMONIA IN AN ADULT, × 850 and reduced.
An air vesicle containing organized tissue in a zone of peribronchitic pneumonia.

The portions of lung which are not hepatized are congested and œdematous. The cavities of the vesicles are diminished by the enlarged capillaries, the epithelium is swollen, and in many vesicles a few pus or epithelial cells are to be found.

Such a broncho-pneumonia differs from the ordinary lobar pneumonia very decidedly. The inflammatory process is not a superficial one, resulting only in filling the bronchi and air spaces with inflammatory products, but it affects also the tissue of the lung, infiltrating the walls of the bronchi and of the air spaces.

This interstitial character of the inflammation seems to be the reason why the disease is often protracted and sometimes succeeded by a chronic inflammation. This chronic condition we will call "*Persistent Broncho-pneumonia.*"

The original acute broncho-pneumonia is succeeded by a chronic inflammation involving especially the interstitial tissue.

This inflammation may involve only some of the smaller bronchi and small zones of vesicles around them, and then a section of the lung will seem to be studded with fibrous nodules. Or all the bronchi of some part of the lung will be inflamed, the peribronchitic zones of pneumonia will become continuous, and so part of a lobe or an entire lobe becomes converted into a dense mass of connective tissue. The air vesicles are obliterated by the new connective tissue, the interlobular septa and the pulmonary pleura are thickened (Fig. 122), and the inflamed bronchi may be dilated. The blood-vessels, however, are, for the most part, not obliterated, so that the lung does not become necrotic or degenerated, although occasionally areas of cheesy degeneration exist.

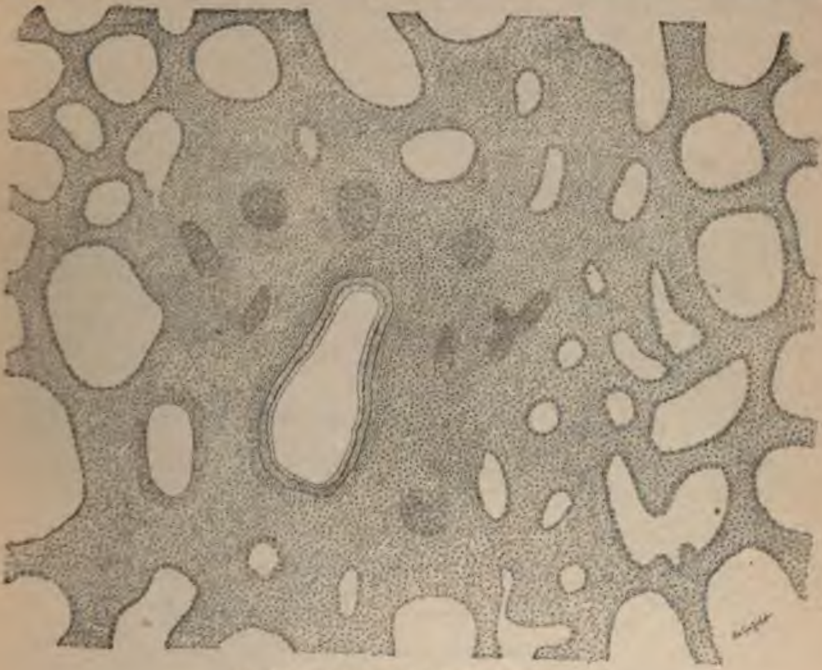

FIG. 122.—PERSISTENT BRONCHO-PNEUMONIA.

III. Secondary and Complicating Pneumonia.

Inflammation of the lungs occurs frequently as a complicating condition with lesions of the brain and spinal cord, with pyæmia, with the continued fevers, after injuries and surgical operations, and in patients who are confined to bed for a long time from any cause.

The pneumonia developed in these cases may follow one of two different types.

1. Part of the lung, usually the posterior portion, is congested, leathery, only partly aërated, and mottled by irregular patches of red or gray hepatization which have no relation to the bronchi. In the hepatized portions of lung, the air spaces are filled with pus and fibrin.

2. The inflammation has the characters of a broncho-pneumonia. The small bronchi are filled with pus, their epithelium is altered, their walls are infiltrated with pus, and around each bronchus is a zone of air vesicles filled with pus and fibrin. The lung is mottled with little whitish nodules, corresponding to the bronchi and the peribronchitic zones, and between these there may be a diffuse hepatization.

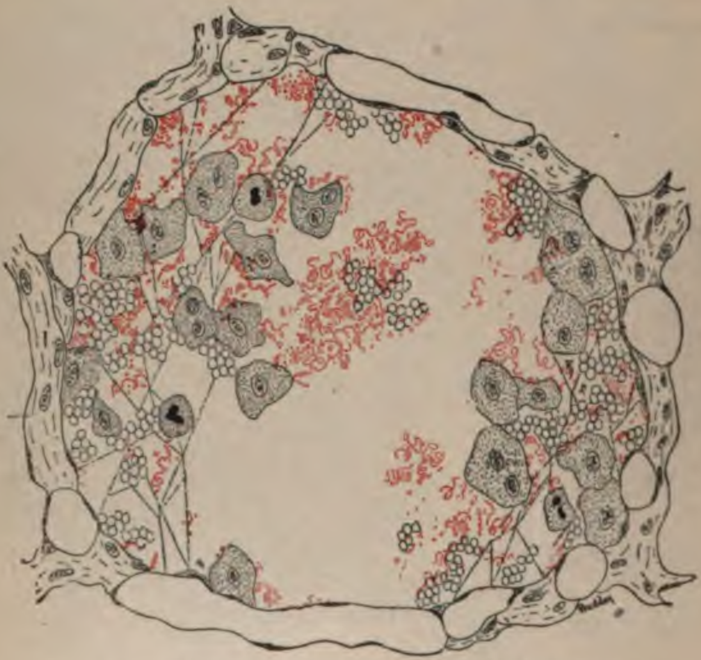

FIG. 123.—LOBULAR PNEUMONIA IN A CHILD, COMPLICATING DIPHTHERIA.

Air vesicle showing inflammatory products and large numbers of bacteria (streptococci) stained red with fuchsin.

In children suffering from diphtheria, with pseudo-membranes containing pathogenic bacteria in the fauces and upper air passages, a secondary pneumonia may apparently occur as the result of the entrance into the lung spaces of the germs from above (Fig. 123).

IV. The Pneumonia of Heart Disease.

Lesions of the aortic and mitral valves, and dilatation of the left ventricle, often produce a diffuse, chronic inflammation of both lungs of a peculiar character. This condition is often called pigment induration, or brown induration, but it is really a chronic pneumonia.

The lungs are diminished in size, and of a peculiar yellowish-pink color, mottled with spots of black or brown pigment. They are not congested, but are of a dry, leathery consistence; or portions of them may be in the condition of a smooth red hepatization. The appearance of these lungs may be modified by the presence of hæmorrhagic infarctions by the pre-existence of emphysema, or by œdema.

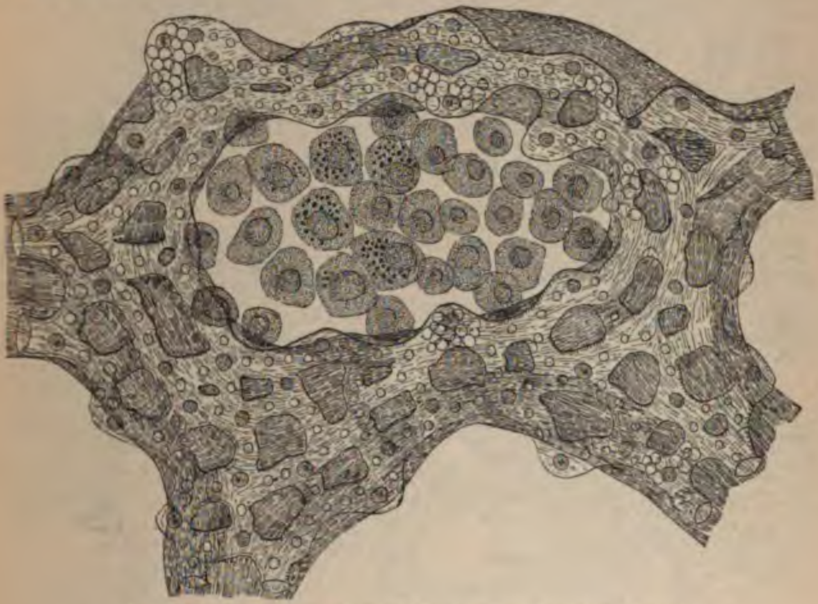

FIG 124.—THE PNEUMONIA OF HEART DISEASE, × 850 and reduced.
An air vesicle from a child's lung.

Minute examination of these lungs shows four separate pathological conditions.

1. A change in the capillaries in the walls of the air spaces. These capillaries are dilated and tortuous, so that they project into the cavities of the vesicles. The degree of the dilatation varies in the different lungs; in some it is very marked, in others but light.

2. A thickening of the walls of the air spaces, due partly to the dilatation of the capillaries, partly to a growth of smooth muscle, and partly to a growth of connective tissue. The degree of the thickening varies very much in different cases.

3. A formation of black or brown pigment in the shape of granules and small masses. This is deposited in the walls of the vesicles, in the interstitial connective tissue, and in the new cells within the vesicles.

4. A formation of cells within the air spaces. The walls of the vesicles are coated with a layer of flat, nucleated cells. Similar cells, or swollen and granular cells, are present in the cavities of the vesicles (Fig. 124). If these cells are numerous, the cavities of the vesicles are filled, and there results a smooth red hepatization.

V. Interstitial Pneumonia.

Moderate degrees of inflammation of the lung, with the production of interstitial connective tissue, are common. Such a development of interstitial pneumonia as to constitute a disease by itself is comparatively rare.

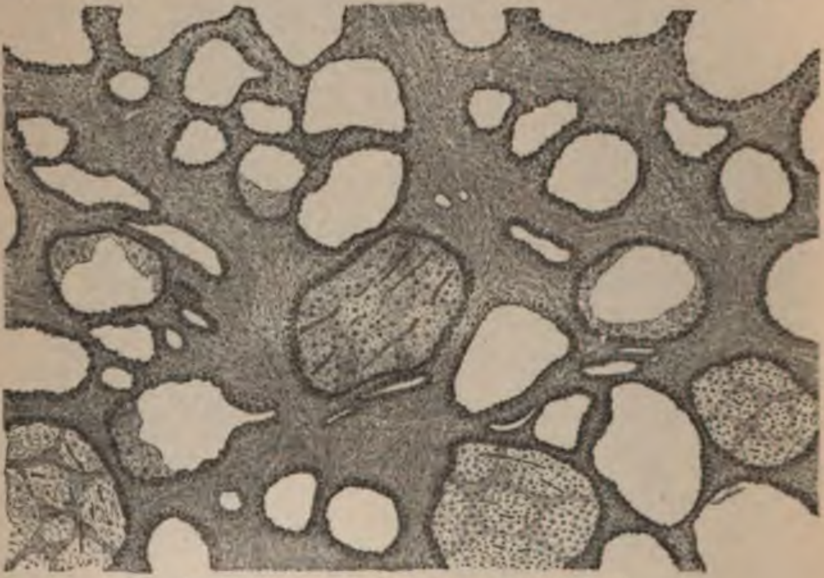

FIG. 125.—INTERSTITIAL PNEUMONIA, × 90 and reduced.
From a case of chronic phthisis.

Causes.—Well-marked interstitial pneumonia follows attacks of broncho-pneumonia; severe attacks of pleurisy which leave behind extensive adhesions; chronic bronchitis; and the inhalation of the dust of coal or stone.

Lesions.—The inflammatory changes are regularly confined to one lung, except in the cases of inhalation pneumonia. This lung is thickly covered with old adhesions, its pulmonary pleura is thickened; the lung itself is small. Bands of dense fibrous tissue extend from the pleura into the lung. There is a growth of connective tissue in the septa between the lobules, around the bronchi and the blood-vessels, and in the walls of the air spaces (Fig. 125). The cavities of the air spaces are compressed and obliterated. The mucous membrane of the bronchi is the seat of chronic catarrhal inflammation. The walls of the bronchi may be thickened and their lumen narrowed; or their walls may be thinned with the formation of bronchiectasiæ.

The other lung is large and emphysematous.

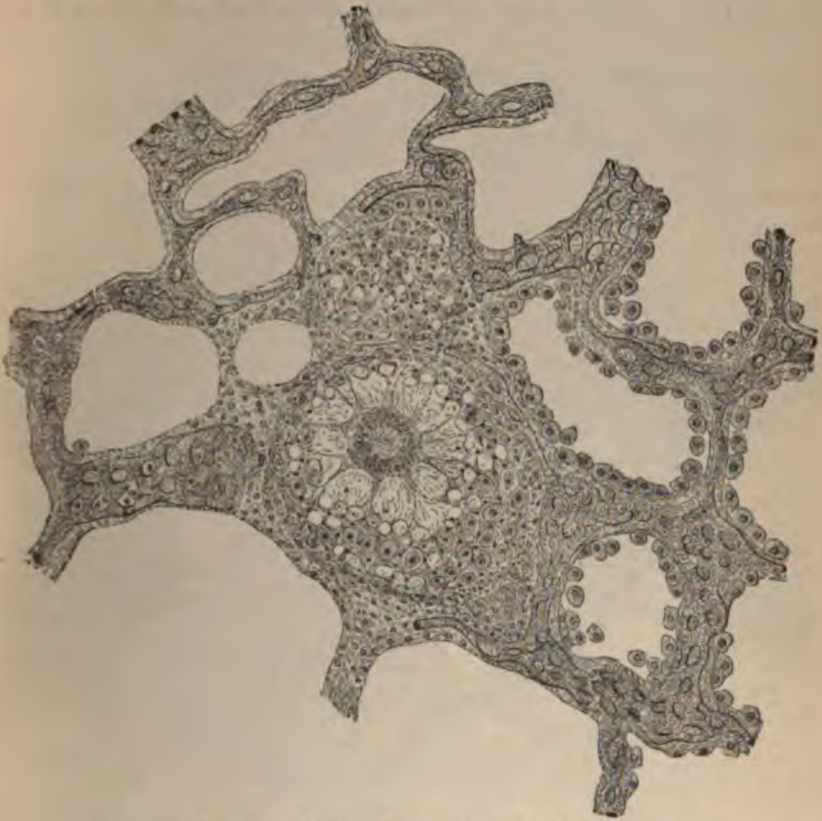

FIG. 126.—A MILIARY TUBERCLE, × 300 and reduced.

Involving only two air vesicles, of which the walls are infiltrated and the cavities filled with tubercle tissue.

Suppurative interstitial pneumonia is sometimes produced in cases of septicæmia. The pulmonary pleura is coated with fibrin, the bronchi contain pus, portions of the lung are hepatized, and the interlobular septa are infiltrated with pus.

VI. Tubercular Pneumonia.

The lungs may become the seat of tubercular inflammation as part of the lesions of an acute general tuberculosis, or they may be affected by localized tubercular inflammations.

The localized tubercular inflammations may conveniently be described under the names of: Acute miliary tuberculosis; Subacute miliary tuberculosis; Chronic miliary tuberculosis; Acute phthisis and Chronic phthisis.

Acute Miliary Tuberculosis.—The acute development of miliary tubercles in the lungs is usually only part of general tuberculosis, although the lesion may be most extensive in the lungs.

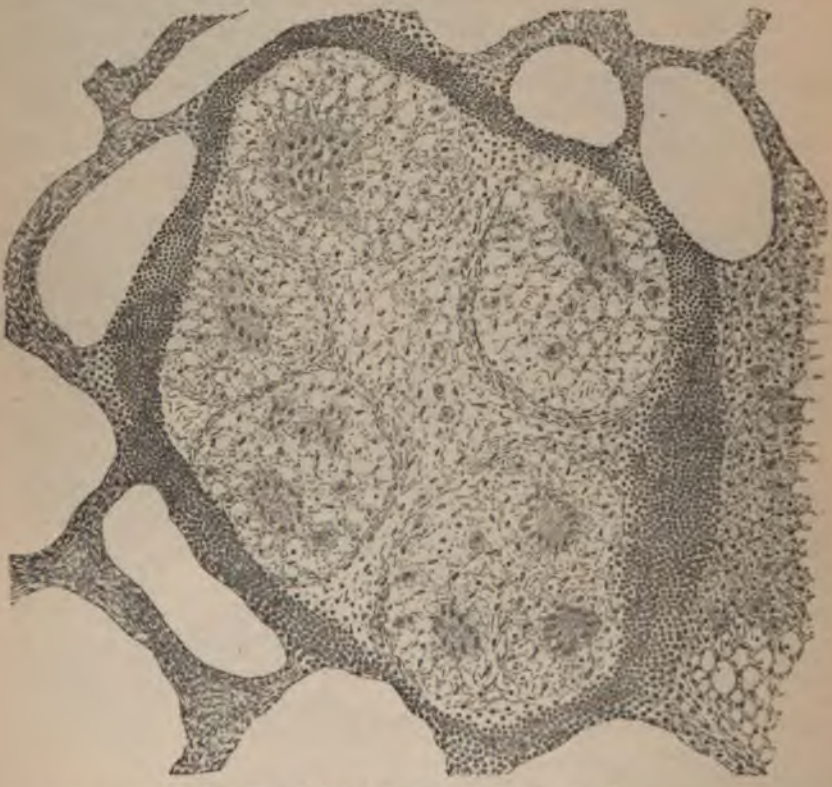

FIG. 127.—A MILIARY TUBERCLE, × 330 and reduced.

Formed of several air vesicles filled with tubercle tissue and surrounded by a zone of tissue resembling granulation tissue.

Both lungs are usually involved, but the distribution, number, size, and character of the miliary tubercles differ in different cases.

The larger bronchi are the seat of catarrhal inflammation; the lung tissue is congested; the air spaces contain epithelium, pus, and fibrin in small quantity.

The tubercles are found in the parenchyma of the lung, in the connective tissue forming the septa, along and in the walls of the bronchi and blood-vessels, and in the pulmonary pleura.

They are scattered singly through the lungs, or aggregated in groups. They may be separated by considerable interspaces, or so close together that the lung is rendered nearly solid. Some are so small and transparent that they can hardly be seen with the naked eye; others are larger and more opaque. In children's lungs, large masses are found of the same structure as miliary tubercles.

When we examine miliary tubercles as they exist in different lungs, it becomes evident that they are not all of the same structure. We find:

1. Miliary tubercles composed entirely of amorphous granular matter, with a few shrunken cells and an external zone of pus cells. These cannot be said to have any definite anatomical structure. In some of them, the outlines of the walls of the air spaces are still visible; in others, they are lost in the cheesy mass.

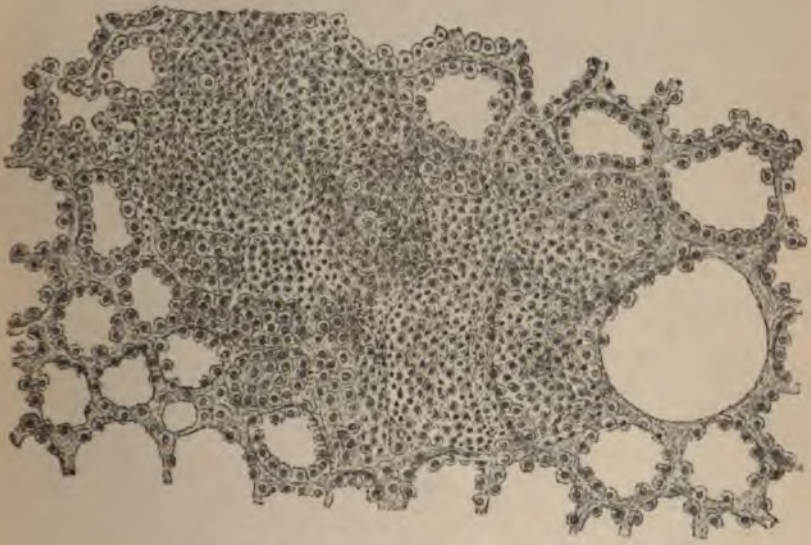

FIG. 128.—A MILIARY TUBERCLE, × 300 and reduced.
Formed of a number of air vesicles, some containing tubercle tissue, others pus and epithelium.

2. Miliary tubercles composed of a group of air spaces of which the walls are infiltrated and the cavities filled (Fig. 126). The walls of the air spaces are infiltrated with granulation tissue or tubercle tissue; as the infiltration progresses, the blood-vessels are obliterated. Such an infiltration may involve symmetrically the whole of the wall of an air space, or only a portion of the wall. The cavities of the air spaces are filled with tubercle tissue, or with epithelium, fibrin, and pus.

In some of these tubercles, the tubercle tissue, both in the walls of the air spaces and in their cavities, is well developed (Fig. 127); then they look like little tumors replacing the lung tissue. In others, the outlines of the walls of the vesicles are preserved, granulation tissue predominates, the cavities of the vesicles contain pus, epithelium, fibrin, and less tubercle tissue (Fig. 128); then the tubercles look like little areas of a composite hepatization.

In adults such tubercles are small, but in children they may reach a large size.

3. Miliary tubercles, formed by the infiltration of the wall of a bronchiole or air passage with tubercle tissue or granulation tissue. This infiltration is apt to involve only one side of the bronchiole or air passage. It may be confined to this or it may extend to the walls of the adjacent vesicles. These vesicles may remain empty, they may be dilated, or they may be filled with tubercle tissue or with epithelium, fibrin, and pus.

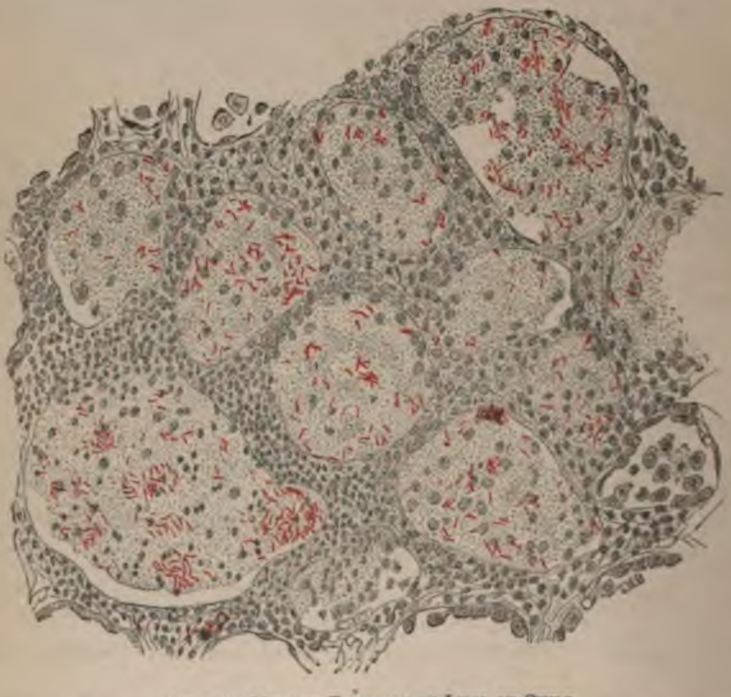

FIG. 129.—MILIARY TUBERCLE IN LUNG OF CHILD.

Showing the Bacillus tuberculosis—stained with fuchsin—in the contents of the air vesicles and in their thickened walls. (The size of the bacilli relative to other elements is slightly exaggerated.)

4. Miliary tubercles, formed by the infiltration of the walls of small bronchi with tubercle tissue or granulation tissue. The infiltration often extends to the surrounding air spaces so as to form tubercles of large size.

In all these miliary tubercles, there is often cheesy degeneration of the central portions.

Tubercle bacilli can be demonstrated in most of these miliary tubercles, especially in those which have undergone cheesy degeneration (Fig. 129).

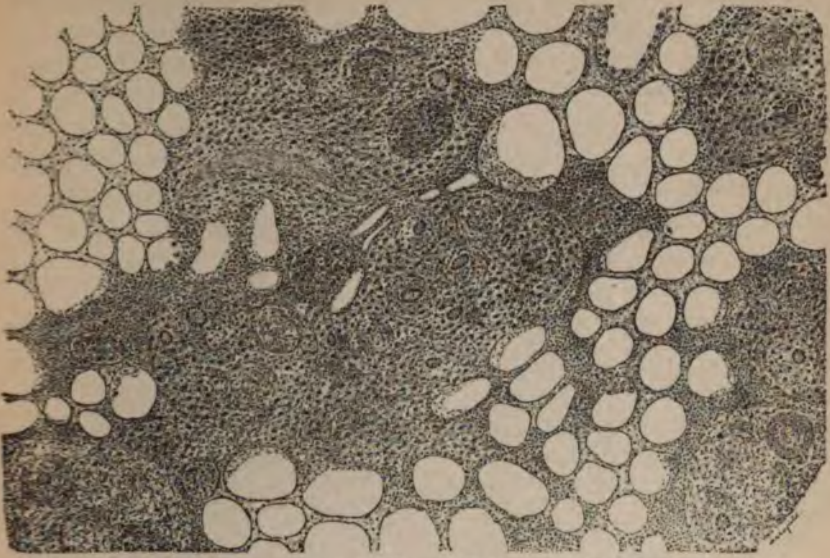

FIG. 130.—AN AGGREGATION OF MILIARY TUBERCLES, × 90 and reduced.

Subacute Miliary Tuberculosis.—The disease involves only the apex of one lung, or one lobe, or portions of both lungs. The inflammation may continue for weeks or months, then stop and the patient recover.

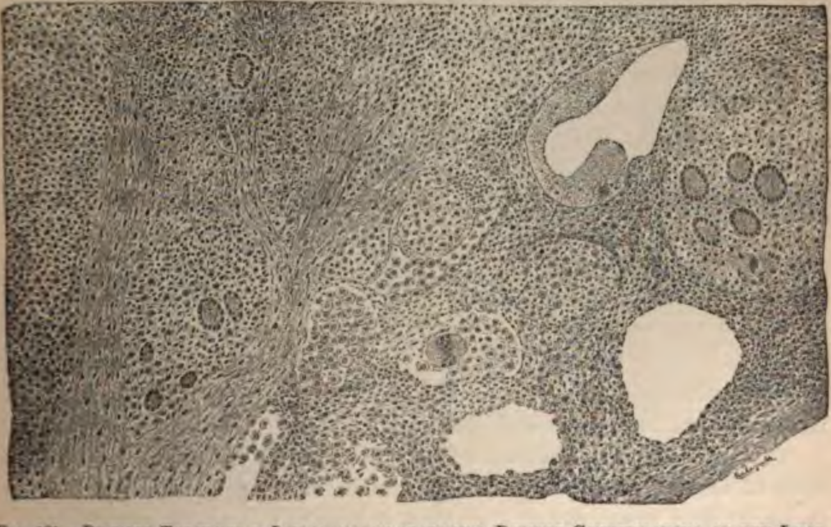

FIG. 131.—DIFFUSE TUBERCULAR INFLAMMATION PRODUCING DIFFUSE CONSOLIDATION OF THE LUNG, × 300 and reduced.

Or the patient may have a number of attacks, from each one of which he recovers. Or the disease may continue, extend, and cause death within a few months. Or it may be succeeded by chronic miliary tuberculosis.

The miliary tubercles are small. Most of them are formed within the air spaces or around the bronchioles. They are composed principally of tubercle tissue or of round-celled tissue. In the portion of lung where the tubercular inflammation is going on, there may also be localized catarrhal bronchitis and pleurisy.

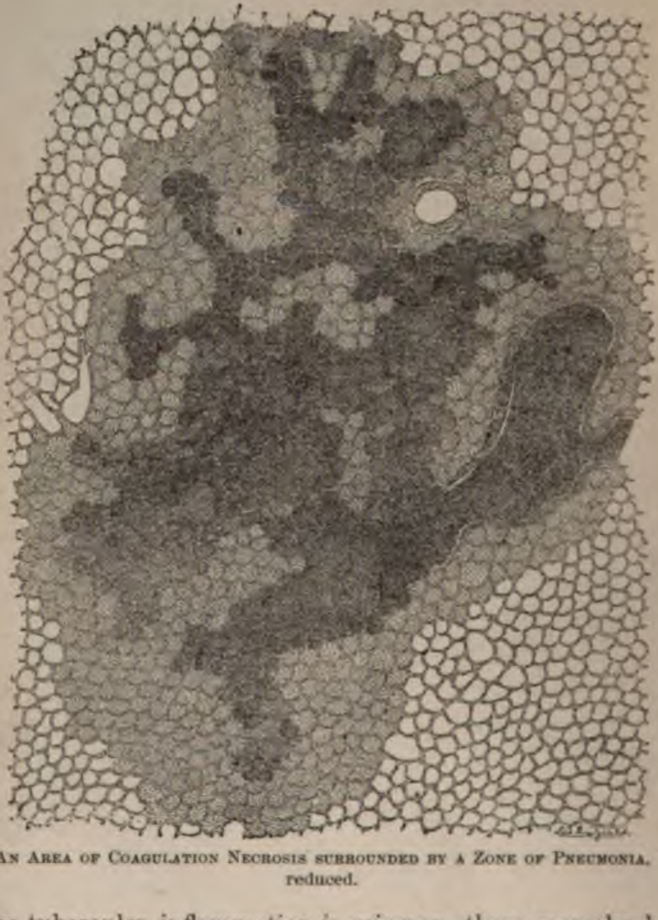

Fig. 132.—An Area of Coagulation Necrosis surrounded by a Zone of Pneumonia. × 40 and reduced.

Chronic Miliary Tuberculosis.—The morbid process begins at the apex of one lung and then slowly extends, either progressively or in attacks, until a large part of the lungs is involved.

In the simplest form of the disease, the only change in the lungs is the formation of miliary tubercles. These tubercles are harder and denser than those found with general tuberculosis or with subacute pulmonary tuberculosis. They are composed of tubercle tissue, or round-celled tissue, or connective tissue, or are in the condition of cheesy degeneration.

Usually, however, in addition to the miliary tubercles there are other changes in the lungs. These additional lesions begin in the same part of the lung where the tubercles are formed, and accompany the development of the tubercles in fresh parts of the lungs.

There may be a localized catarrhal bronchitis.

There may be an inflammation of the walls of the bronchi, with partial destruction of these walls and the formation of cylindrical or sacculated bronchiectasiæ. The walls of the cavities thus formed may be converted into connective tissue, or they may remain suppurating and necrotic.

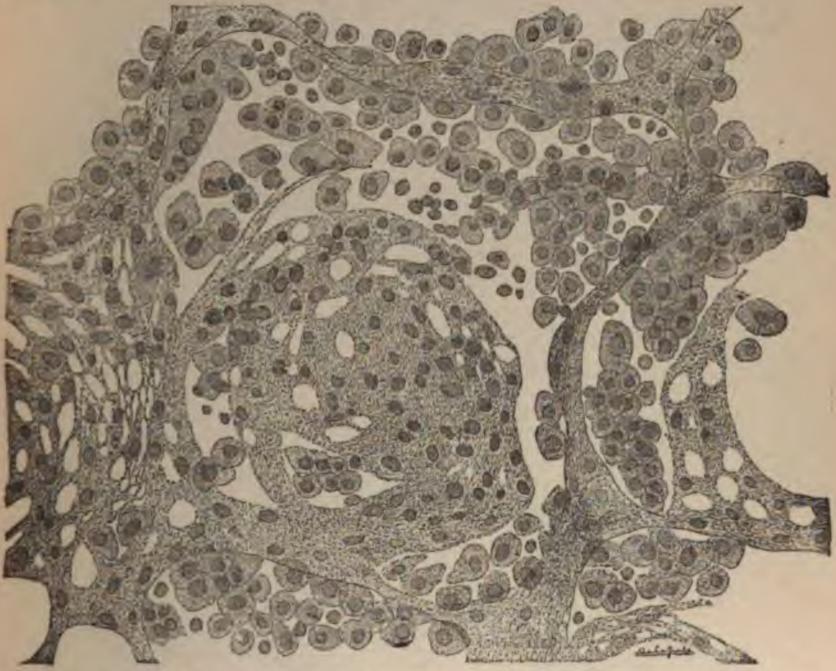

FIG. 133.—TUBERCLE TISSUE AROUND AN AREA OF COAGULATION NECROSIS, × 850 and reduced.

There may be an interstitial pneumonia with the production of new connective tissue, the obliteration of the air spaces, and the consolidation of portions of the lungs.

There may be dilatation of the air spaces of the portions of the lungs which are not consolidated (Fig. 130).

There may be thickening of the pulmonary and costal pleura, with connective-tissue adhesions.

While the morbid process begins as a localized tubercular inflammation of the lungs, and often retains throughout this local character, yet it may also happen that from this local lesion other parts of the body may be infected. Tubercular laryngitis, and tubercular inflammation of the solitary and agminated glands of the small intestine, often complicate the pulmonary lesion, and sometimes even acute general tuberculosis is produced.

Acute Pulmonary Phthisis (Acute consumption; Acute catarrhal phthisis).—This name is used to designate an acute tubercular inflammation of the lungs combined with other non-tubercular inflammatory changes.

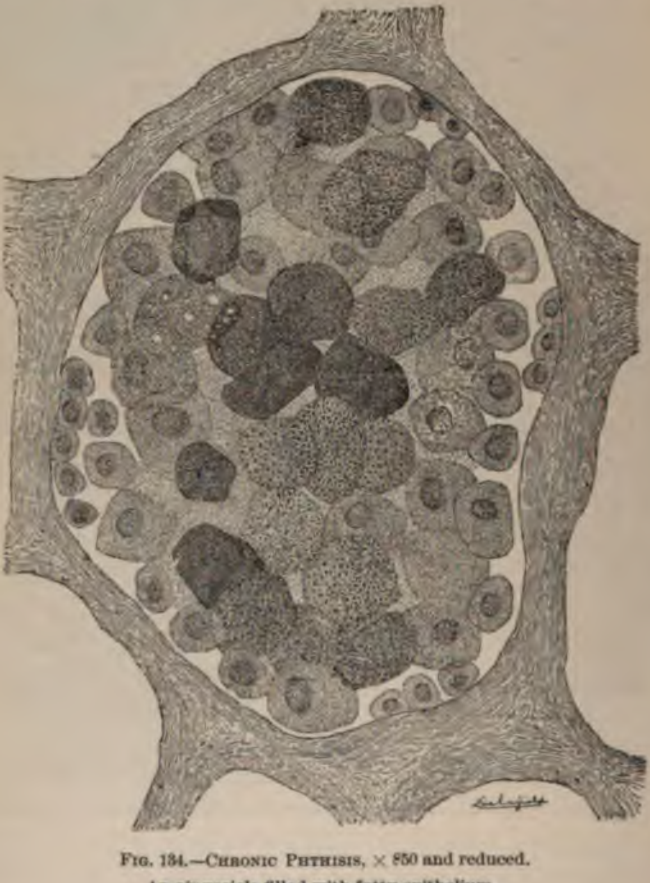

FIG. 134.—CHRONIC PHTHISIS, × 850 and reduced.
An air vesicle filled with fatty epithelium.

1. In one or more lobes there may be miliary tubercles and a diffuse consolidation, due to the filling of the air spaces with fibrin, pus, and epithelium.

2. There is a tubercular broncho-pneumonia involving the smaller

bronchi of one or more lobes, and zones of air spaces surrounding these bronchi. The walls of the bronchi and of the surrounding air vesicles are infiltrated with tubercle tissue.

3. Besides the tubercular inflammation of the walls of the bronchi and of the air spaces surrounding them, there are a general catarrhal bronchitis, diffuse consolidation of the lung by the filling of the air spaces with fibrin, pus, and epithelium, and fibrin on the pulmonary pleura.

4. Besides the tubercular broncho-pneumonia, the diffuse consolidation, and the pleurisy, there are small or large portions of dead lung tissue in the condition of coagulation necrosis (Fig. 132). These areas pass into the condition of cheesy degeneration, and are surrounded by zones of tubercle tissue or of round-celled tissue; or they soften and form cavities which are in communication with the bronchi.

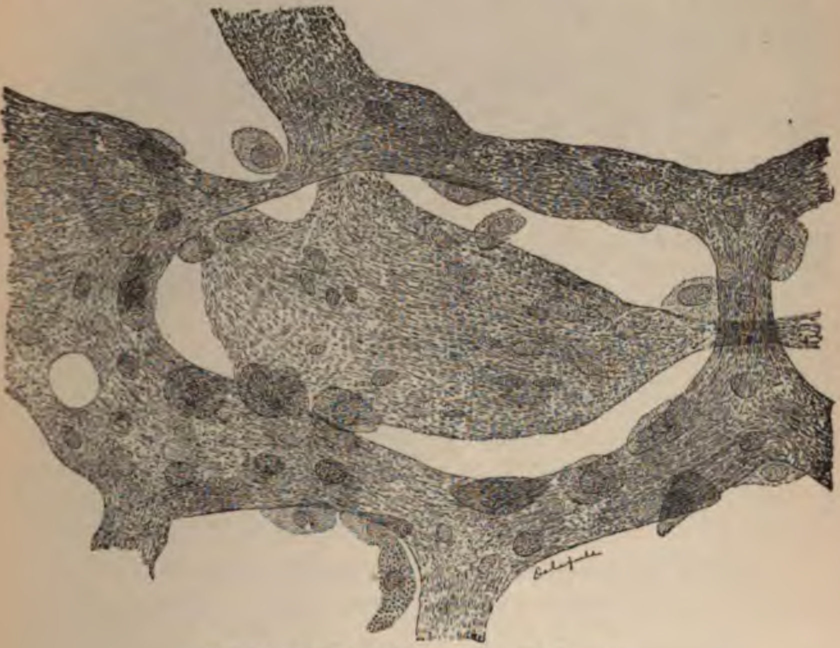

FIG. 135.—CHRONIC PHTHISIS, × 850 and reduced.
Showing growth of connective tissue within an air vesicle.

5. In addition to the lesions just mentioned, the walls of the bronchi are so changed by the tubercular inflammation that cylindrical or sacculated bronchiectasiæ are formed.

Chronic Phthisis.—The lesions are of the same nature as those of acute phthisis, but are modified by the long continuance of the inflammation.

1. The air spaces:

(*a*) The air spaces are filled with swollen and fatty epithelium (Fig. 134), or with fibrin and pus, while their walls are unchanged and their blood-vessels remain pervious.

(*b*) The air spaces are filled and distended with compact fibrin and shrivelled pus and epithelium. Their walls are compressed and thin, or thickened and infiltrated with cells. The blood-vessels can be only very imperfectly injected. This condition may be succeeded by complete cheesy degeneration.

(*c*) The walls of the vesicles are thickened, their cavities are filled with new connective tissue often containing new vessels (Figs. 135 and 136). This new connective tissue may look like an outgrowth from the wall of the vesicle, or as if it was formed free in its cavity.

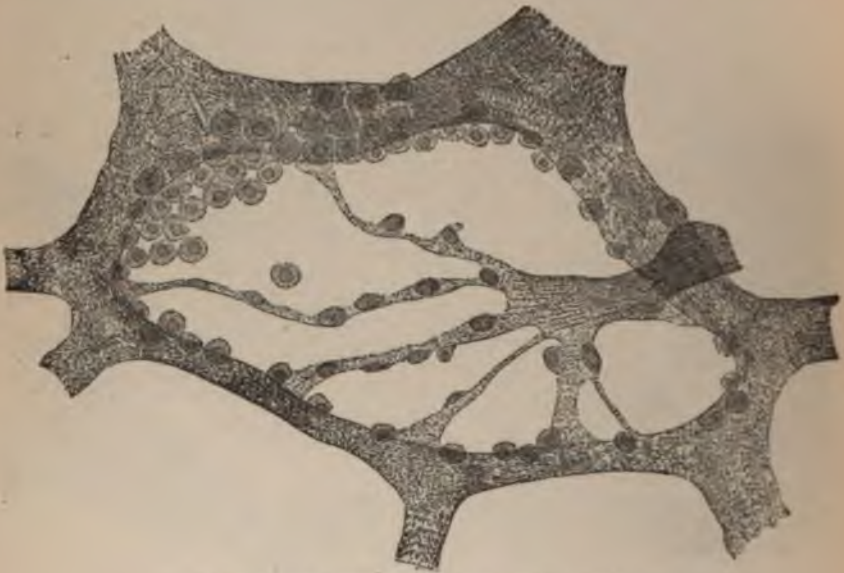

FIG. 136.—CHRONIC PHTHISIS, ×850 and reduced.
Showing growth of connective tissue within an air vesicle.

(*d*) There is a diffuse interstitial growth of fibrous tissue and granulation tissue in the walls of the air spaces, the bronchi and the blood-vessels, and in the septa. By this new tissue the air spaces are compressed and deformed or completely obliterated (Fig. 137).

2. The nodules:

These, as in acute phthisis, consist of areas of coagulation necrosis, peribronchitic nodules, and miliary tubercles.

The tubercles may preserve their characteristic structure, or undergo cheesy degeneration, or be changed into fibrous tissue (Fig. 138).

The areas of coagulation necrosis undergo cheesy degeneration, or soften and form cavities. They are surrounded by tubercle tissue, or granulation tissue, or connective tissue.

The peribronchitic nodules are much the same as in acute phthisis.

3. The bronchi:

The changes in the bronchi in chronic phthisis form a very important part of the morbid process.

(*a*) The larger bronchi may be the seat of a chronic catarrhal inflammation, accompanied by the production of large quantities of mucus and pus.

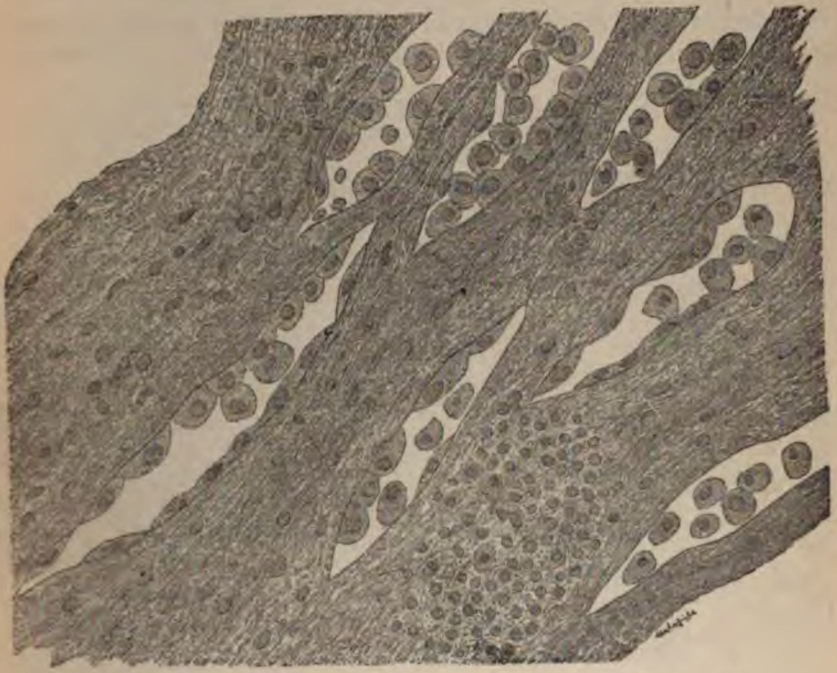

FIG. 137.—INTERSTITIAL PNEUMONIA OF CHRONIC PHTHISIS, × 850 and reduced.

(*b*) The bronchi of all sizes may be inflamed, with the production of new cells in their walls, in addition to the inflammatory changes of their inner surfaces. Such a cellular infiltration of the walls of the bronchi is often followed by dilatation—either fusiform or sacculated.

(*c*) Tubercle granula and granulation tissue are found in the walls of the bronchi. These tissues may degenerate, soften, and thus form ulcers.

(*d*) The entire thickness of the wall of a bronchus may become the seat of inflammation of a peculiar character. The surface of the mucous membrane is coated with pus, the epithelial layer can no longer be seen, the wall of the bronchus is infiltrated with cells. The inflammatory products undergo cheesy degeneration, so that we find the inner surface of the bronchus coated with cheesy matter, while its wall is also changed into cheesy matter. Such a condition of the bronchus is usually followed by sacculated dilatation.

The cavities of chronic phthisis, therefore, are formed by the dilatation of inflamed bronchi, by the softening of areas of coagulation necrosis, or by the combination of both these processes.

When cavities are once formed, they are apt to continue and to become larger as the disease goes on. Their walls may be converted into granulation tissue, which ulcerates in some places and proliferates in others; or portions of the wall become necrotic; or all active processes cease and the wall of the cavity is formed of new connective tissue. The lung tissue between the cavities becomes compressed and altered in various ways. As the cavities increase in size, they touch and open into each other. In this way large portions of the lung may be converted into a dense mass honeycombed with cavities.

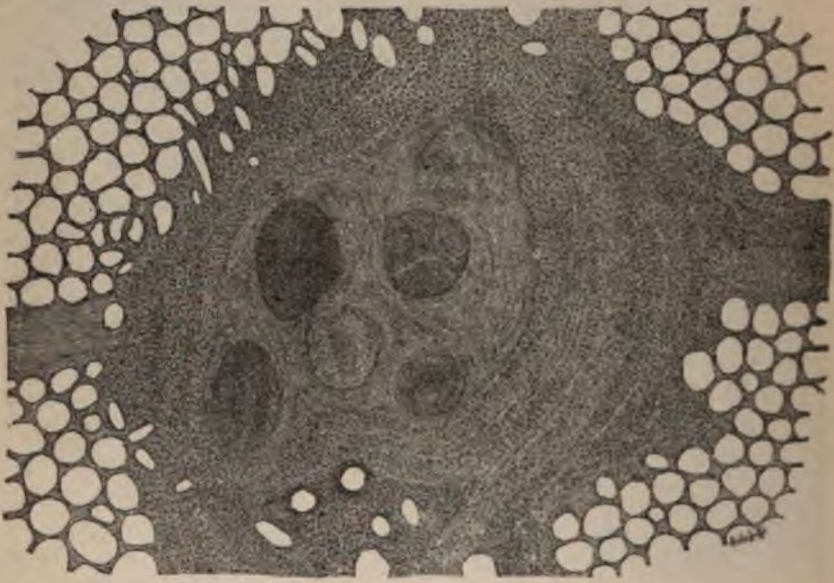

FIG. 138.—AN OLD MILIARY TUBERCLE CONVERTED INTO FIBROUS TISSUE, × 90 and reduced.

VII. *Syphilitic Pneumonia.*

Persons suffering from inherited or acquired syphilis sometimes develop inflammations of the lungs which seem to be due to the syphilitic infection. The lungs may then be affected in several different ways.

1. There is an interstitial pneumonia beginning around the larger bronchi and blood-vessels at the root of the lung, and extending to the walls of the air spaces and interstitial connective tissue, so that the central portions of one or both lungs are converted into a dense mass of connective tissue (Fig. 139).

2. There is an interstitial pneumonia, with the formation of gummy tumors.

3. There is an inflammation of the wall of the trachea and of the larger bronchi. There are ulcers in the mucous membrane, their walls are very much thickened, and their cavities are narrowed or dilated.

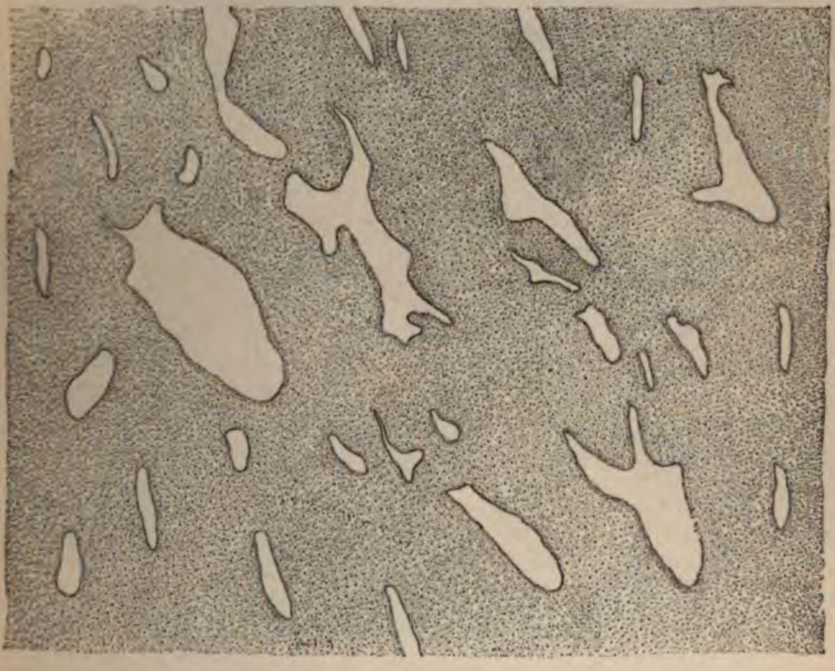

FIG. 139.—INTERSTITIAL SYPHILITIC PNEUMONIA, × 170 and reduced.

4. There are circumscribed areas of interstitial inflammation around the smaller bronchi, forming small, hard peribronchitic nodules.

5. There is a diffuse hepatization, involving lobules or an entire lobe. The affected portion of the lung is red or white or grayish. The walls of the air vesicles are infiltrated with cells, and their cavities are filled with epithelial cells.

6. There may be a broncho-pneumonia, like the ordinary broncho-pneumonia of children ; or a lobar pneumonia, like that of adults.

7. There may be an obliterating endarteritis of branches of the pul-

monary artery, with the formation of white infarctions surrounded by zones of connective tissue.[1]

TUMORS.

Dermoid cysts have been found in the lungs in a few instances.

Fibromata have been described by Rokitansky.

Enchondromata may occur both as primary and secondary tumors. The primary tumors are small and are believed to originate in the cartilages of the bronchi. The secondary tumors often attain a very large size.

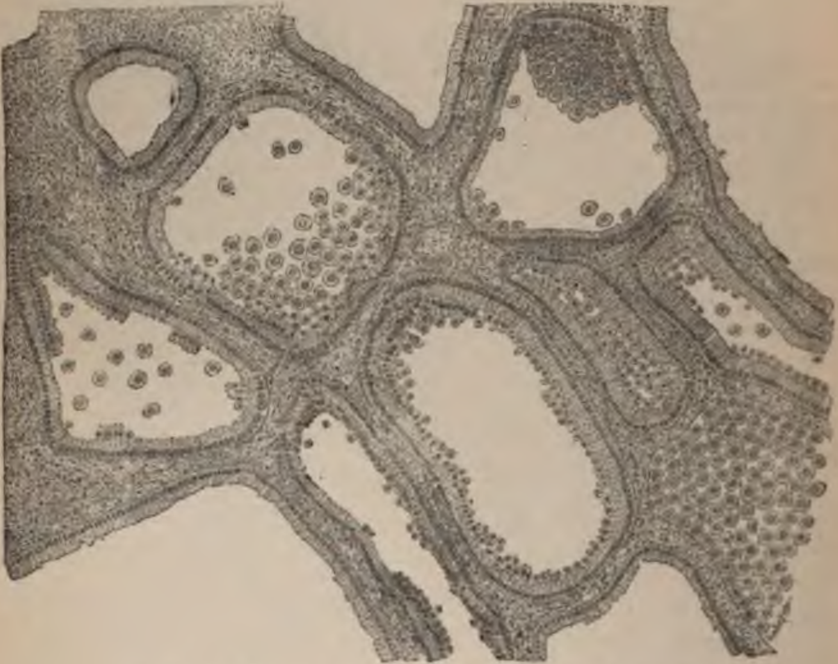

FIG. 140.—PRIMARY CARCINOMA OF THE LUNG, × 300 and reduced.

Osteoma is very rare. A case is described by Luschka.[2]

Sarcomata as secondary tumors are of not infrequent occurrence. A primary adeno-sarcoma is described by Weichselbaum.[3]

Lymphomata are found in cases of leukæmia and pseudo-leukæmia.

Carcinoma as a secondary growth may have the form of nodules or of diffuse infiltration. Primary carcinoma of the lung has been described by a number of authors. The new growth (Fig. 140) is in the form of small nodules surrounded by pneumonia. As the result of the new growth and the pneumonia, a considerable part of both lungs may be rendered solid. The bronchial glands are infiltrated, and there may be secondary nodules in the pleura.

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[1] Hiller, Charité Annalen, 1884, p. 184.

[2] Virch. Arch., x., p. 500.

[3] Virch. Arch., 85, p. 559.

The new growth seems to originate in the air spaces. Their walls are thickened, their cavities are lined with cylindrical epithelium, or filled with cylindrical and polygonal cells, some of which undergo colloid degeneration.

Or it may originate in the small bronchi.[1]

PARASITES.

Echinococci occur in the lungs in their ordinary cystic form. The sacs may suppurate and discharge through the pleura, the bronchi, the wall of the chest, or the diaphragm.

In bronchiectasiæ and in gangrenous cavities in the lungs, vegetable parasites of various kinds have been described—both moulds and bacteria.[2]

The Bacillus tuberculosis is regularly found in the walls and contents of cavities in acute and chronic phthisis, sometimes in enormous numbers. They are also often present in great numbers in the nodules of tubercular inflammation, particularly when these are softening and beginning to break down to form cavities (see Tuberculosis).

THE MEDIASTINUM.

The anterior mediastinum is situated in front of the pericardium, between it and the sternum. At its superior part, the two layers of pleuræ separate somewhat to inclose the vestiges of the thymus gland; behind the second piece of the sternum they are in contact, but below this the left pleura recedes from its fellow towards the left side, leaving an angular space of some breadth. The triangularis sterni muscle bounds this space in front.

The posterior mediastinum, stretching from the pericardium to the bodies of the vertebræ, incloses between its layers the lower part of the windpipe and gullet, the thoracic duct, the descending aorta, the azygous vein, the pneumogastric nerve, and some lymphatic glands.

INFLAMMATION.

Suppurative inflammation may occur either in the anterior or posterior mediastinum. It may be caused by fractures, caries, or necrosis of the sternum and vertebræ, by perforation of the œsophagus, by suppuration of the lymphatic glands, by pleurisy, or may occur without discoverable cause.

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[1] Virch. Arch., Bd. 83, p. 77.

[2] Virch. Arch., Bd. 66, p. 330.

The pus may infiltrate the connective tissue, or may form abscesses which may attain a large size. The inflammation may extend to the pleura or the pericardium; the abscesses may displace the heart, the lungs, or the sternum; or they may perforate through the skin into a pleural cavity, the œsophagus, the trachea, or a bronchus.

TUMORS.[1]

The most common form of new growth in the mediastinum is that known by the names of lymphoma, lympho-sarcoma, and lymph-adenoma.

These tumors are confined to the mediastinum, or they are associated with similar growths in other parts of the body in the disease called "pseudo-leukæmia."

Persons between the ages of twenty and thirty years seem to be the most liable to the growth, but it is also not uncommon in children.

The growth begins in the lymphatic glands in the mediastinum, and at the root of the lung. It increases at first slowly, then more rapidly, and gradually infiltrates the adjoining tissues. In this way, the walls of the trachea, bronchi, and aorta, the pericardium, the pleura, and the lung, become infiltrated with the growth. The tumor also compresses the surrounding organs.

The growth is composed of a connective-tissue stroma infiltrated with small round cells, the relative quantity of cells and stroma varying in the different cases.

Besides this form of tumor, there may also occur in the mediastinum tumors similar to those which grow in the pleura and behind the peritoneum, tumors which resemble both the sarcomata and carcinomata and which are difficult to classify.

Teratoma Myomatodes.—Under this name Virchow describes a very remarkable tumor. It grew rapidly in a man, aged twenty-two, of good constitution and physical development. The right pleural cavity was nearly filled with a solid growth. There were similar growths, of smaller size, on the left rib, in the liver, spleen, and kidneys. The mediastinal tumor consisted of two portions, a larger and a smaller. The larger portion consisted of solid tissue, in which were a few cavities. It was very vascular. Part of it was composed of dense fibrous tissue. The greater portion, however, consisted of loose connective-tissue fibres, of fusiform cells, and of large, many-nucleated cells. Some of the fusiform cells were small and of the usual appearance; but many were large, and presented striations like those of young voluntary muscle. The smaller portion of the tumor looked like a multilocular cystoid. The cysts varied much in size, and contained serum, colloid matter, and blood. Some of the cysts contained a thick, white material, in which were hairs, epidermis scales, ciliated epithelium, and cholestearin. In the fibrous tissue about these cysts were pieces of hyaline cartilage. Near the surface of the tumor the tissue consisted of a fibro-cellular stroma forming alveoli filled with epithelium. The tumor on the third left rib consisted of connective-tissue fibres and cells, muscle cells, cysts, alveoli filled with epithelium, and a small portion of tissue resembling fœtal lung.

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[1] Consult *Hare*, "Tumors of the Mediastinum," Philadelphia, 1889.

THE VASCULAR SYSTEM.

THE PERICARDIUM.

INJURIES.

The pericardium may be wounded by penetrating weapons, by gunshot wounds, and by fragments of bone. It may be ruptured by severe contusions of the thorax, and by rapid extravasation of blood into the pericardial sac.

Perforations may be produced by empyema, by mediastinal abscesses, by abscesses of the chest wall and of the liver, by aneurisms of the aorta, and by suppurative inflammation of the pericardium.

DROPSY.

In most post-mortems, we find a little serum, from one-half ounce to one ounce, in the pericardial sac. This serum is usually clear and of a light yellow color; if decomposition has commenced, it may be of a reddish color, or it may be slightly turbid from the falling-off of the pericardial epithelium.

Large accumulations of serum are found as part of general dropsy from heart disease, kidney disease, etc. The serum is clear and of a light yellow color. Hydro-pericardium is usually moderate in comparison with the accumulations of serum in the other serous cavities; sometimes, however, there is a very large amount of serum, which hinders the movements and interferes with the nourishment of the heart.

HÆMORRHAGE.

Extravasations of blood in the cavity of the pericardium are produced by wounds and rupture of the heart, rupture of the aorta and of aneurisms, and occur with pericarditis Small extravasations in the substance of the pericardium are found with scurvy, purpura, fevers, etc.

PNEUMONATOSIS.

Air or gas in the pericardium is sometimes found as a post-mortem appearance, accompanied with drying of portions of the pericardium.

Wounds or paracentesis of the pericardium; the perforation of ulcers of the stomach, cavities of the lungs, and ulcers of the œsophagus, may admit air into the pericardial cavity. In purulent pericarditis, with foul, decomposing exudation, gases may be evolved.

INFLAMMATION.

Pericarditis is very rarely a primary lesion. It is most frequently associated with rheumatism and Bright's disease, but is also found with pneumonia, pleurisy, phthisis, endocarditis, pyæmia, and may be produced by injuries.

The inflammations of the pericardium resemble those of the pleura. They usually begin acutely or subacutely, but may become chronic. There is a greater disposition to the escape of blood from the vessels than in pleurisy, so that the inflammatory products are often mixed with blood. The inflammatory process usually begins at the base of the heart and from there extends over the rest of the pericardium.

Exudative Pericarditis.

We may distinguish:

1. *Pericarditis with the Production of Fibrin.*—In the milder examples of this form of pericarditis, the pericardium is congested, or also studded with minute hæmorrhages, its surface is roughened by the deposition of a thin layer of fibrin. In the more severe cases, the entire surface of the pericardium is covered with a thick layer of fibrin, and there are fibrinous adhesions between the visceral and parietal pericardium. If the inflammation continues for any length of time, the pericardium itself becomes thickened and infiltrated with cells, and the wall of the heart may also undergo inflammatory changes.

If the patient recovers, the fibrin may be absorbed and the pericardium return to its normal condition. Or, instead of this, as the fibrin disappears there is a growth of new connective tissue which forms permanent thickenings and adhesions of the pericardium, which may afterwards become calcified.

2. *Pericarditis with the Production of Fibrin and a good deal of Serum.*—In these cases, the pericardium is coated with fibrin, but, in addition, there is a large effusion of serum into the pericardial sac. This serum accumulates at first between the floor of the pericardium and the lower surface of the heart, and, as it increases, distends the pericardial sac in all directions, pushing the heart upward and forward. The pericardial sac may be so much distended as to compress the trachea, the left bronchus, the œsophagus, or the aorta. If the patients recover, the serum is absorbed, and permanent adhesions and thickenings are left.

3. *Pericarditis with the Production of Fibrin, Serum, and a good deal*

of Pus.—This variety may have the purulent character from the outset, or it may begin as one of the forms just described, and afterwards assume the purulent character. These latter cases are apt to run a chronic course.

In the chronic cases, the pericardial sac contains a large amount of purulent serum. The pericardium is coated with fibrin and is itself thickened and infiltrated with cells. The walls of the heart may be the seat of interstitial myocarditis. In some cases, the products of inflammation undergo putrefactive changes; in some cases, the serum is absorbed and the fibrin and pus undergo cheesy degeneration; in some cases, extensive connective-tissue adhesions and calcific plates are formed.

4. *Tubercular Pericarditis.*—This lesion may occur by itself, but is apt to be associated with tubercular inflammation in the vicinity of the heart. There may be miliary tubercles scattered diffusely, or limited to certain regions in the pericardium, which is otherwise little changed. Not infrequently, however, there is a considerable thickening of the pericardium, either visceral or parietal, or both.

In such cases, the new-formed tissue consists of fibrous tissue and of tubercle tissue which has undergone extensive cheesy degeneration. The thickened visceral and parietal pericardium are often more or less grown together, so that the pericardial sac may be partially or almost completely obliterated. A simple inflammatory exudation of varying form often accompanies the tubercular process.

TUMORS.

Fibromata sometimes are developed in the pericardium. They are often of polypoid form, and from atrophy of the pedicle may become free in the pericardial sac.

Sarcomata and *carcinomata* occur as secondary growths either from continuous infiltration or as metastatic tumors.

Cysts of the visceral pericardium have been described.

We have seen a pedunculated cyst containing about six c.c. of clear fluid hanging into the pericardial sac from its attachment near the pulmonary artery. The origin of these cysts is obscure.

Endothelioma.—There may be a growth of flat cells arranged in anastomosing tubules which look like lymphatics, in the pericardium, resembling similar growths in the pleura.

THE HEART.

MALFORMATIONS.

The malformations of the heart are usually closely connected with malformations of the aorta and pulmonary artery. They depend on arrest of, or abnormal, development; on endocarditis, myocarditis, thrombosis, or mechanical causes.

I. The common arterial trunk is only partially, or not at all, separated into aorta and pulmonary artery. The divisions between the heart cavities are at the same time defective.

1. There is one ventricle and no auricle.

2. There is one ventricle and one auricle.

3. There is one ventricle and two auricles; the aorta is alone or incompletely separated from the pulmonary artery.

II. The trunk of the pulmonary artery or of the aorta is stenosed or obliterated, and from the obstruction to the current of blood the development of the septa, between the heart cavities, is prevented.

1. The aorta, at its origin, or in the ascending portion of the arch, is stenosed or closed. The pulmonary artery gives off the descending aorta, and supplies the carotids and subclavians. The foramen ovale remains open, or there is no septum between the auricles. The ventricular septum is also usually defective. The right ventricle is hypertrophied.

2. The pulmonary artery is stenosed or closed. Its branches are supplied by the aorta, through the ductus arteriosus. The ventricular septum is defective, the foramen ovale is open, or the auricular septum defective.

III. The malformation affects the aorta and pulmonary artery after they are more fully developed.

1. There is stenosis of the aorta between the left subclavian and ductus arteriosus, or just at the opening of the ductus arteriosus. The descending aorta is then a continuation of the pulmonary artery.

2. The aorta gives off all its branches from the arch, but the descending aorta is a continuation of the pulmonary artery; or the carotids may spring from the aorta, the subclavians from the pulmonary artery.

3. The vessels are transposed; the pulmonary artery arises from the left, the aorta from the right ventricle; the pulmonary veins empty into the left, the venæ cavæ into the right auricle; or the veins also may be transposed. The septa are defective.

IV. The aorta and pulmonary artery are normal, but the cardiac septa are defective.

1. The foramen ovale remains partly open. This condition may continue through life without giving any trouble.

2. The ductus arteriosus may remain open for many years; this also may cause no disturbance.

3. There is a small or large opening in the ventricular septum. This may give rise to no symptoms, unless disease of the heart or lungs be superadded.

V. Either of the auriculo-ventricular orifices may be entirely closed. The foramen ovale remains open, and the ventricular septum is defective.

VI. The valves of the different orifices of the heart may be absent or defective. The arteries or the ventricles are usually defective at the same time.

The aortic and pulmonary valves may consist of two large or four small leaves, instead of the usual three. The edges of the semilunar valves may be fenestrated. These alterations are usually of no significance.

Generally speaking, the existence of openings between the two auricles or the two ventricles, admitting some admixture of venous and arterial blood, produces no marked change in the circulation. If, however, the passage of the current of venous blood into the right heart is in any way interfered with, the consequences are very serious. Cyanosis is produced, the skin is of a bluish color, the small veins and capillaries are dilated, exudation of serum and hypertrophy of connective tissue take place, especially in the fingers and toes.

Besides the malformations already mentioned, we may find :

Entire absence of the heart.

Abnormal septa and cordæ tendineæ in the heart cavities.

Abnormal shapes of the heart.

Abnormal positions of the heart.

(*a*) There is a smaller or larger defect in the walls of the thorax, so that the heart projects on the outside of the chest; the pericardium is usually absent.

(*b*) The diaphragm is absent, and the heart is in the abdominal cavity.

(*c*) The heart is in some part of the neck or head; this occurs only in fœtuses very much malformed.

(*d*) The heart is transposed, being on the right side.

ABNORMAL SIZE OF THE HEART.

(*a*) The heart may be abnormally large in connection with obstructive anomalies of the great vessels.

(*b*) The heart may be abnormally small (hypoplasia). This most frequently occurs, according to Virchow, in chlorotic individuals and those who are the victims of the hæmorrhagic diathesis. In these cases, the aorta and other large arteries are apt to be unusually small and thin-walled.

Very rarely two more or less perfect hearts are found in the same thorax.

CHANGES IN POSITION.

Changes in the position of the heart are congenital or acquired. The congenital malpositions have already been mentioned.

The acquired malpositions are caused by:

1. Hypertrophy of the heart; its long axis approaches the horizontal direction.

2. Changes in the thoracic viscera. Emphysema of both lungs pushes the heart downward. Emphysema, pleurisy with effusion, or pneumothorax of one side pushes the heart to the other side. Pleurisy or chronic pneumonia, producing retraction of one side of the thorax, draws the heart to that side. New growths, aneurisms, and curvatures of the spine displace the heart in various directions.

3. Changes in the abdomen. Accumulations of fluid and new growths in the abdomen, and tympanites, may push the heart upward.

WOUNDS AND RUPTURES.

Wounds of the heart are produced by penetrating instruments, by bullets, and by fragments of bone. The right ventricle is the more frequently wounded; next, the left; rarely, the auricles.

The wound may penetrate into the cavities of the heart, or only pass partly through its wall, or a bullet or the broken end of a weapon may be imbedded in the wall. If the wound penetrates into a cavity, and is gaping, death follows instantly, and the pericardium is found filled with blood. If the wound be small and oblique, the blood may escape gradually, and death may not ensue for several days. In rare cases, adhesions are formed with the pericardium, and the wound cicatrizes. Wounds which do not penetrate may cause death by the inflammation which they excite, or may cicatrize.

Bullets and foreign bodies may become incapsulated in the heart wall, and remain so for years.

Ruptures of the heart wall occur in various ways:

1. Severe contusions of the thorax may produce rupture, usually of one of the auricles.

2. Spontaneous rupture occurs usually in advanced life. Rupture is most frequent in the left ventricle and, in a considerable proportion of cases, near the apex. There is usually one rupture, but sometimes more. The rupture is usually oblique and larger internally than externally. The heart wall, near the seat of rupture, may be infiltrated with blood, or blood may infiltrate the subpericardial fat. The heart wall may be of normal thickness, or thin; it is usually soft and in a condition of fatty infiltration or degeneration. The rupture very frequently takes place when the patient is quiet. Death may be almost instantaneous or may not ensue for several hours.

Fatty degeneration leading to rupture of the heart may be general, or it is frequently circumscribed and due to obliterating endarteritis, atheroma, thrombosis, or embolus of one of the coronary arteries, whereby a portion of the heart wall is deprived of nourishment and degenerates.

Or rupture of a branch of one of the coronary arteries may induce rupture of the heart wall. Acute and chronic myocarditis, with or without the formation of abscess or cardiac aneurism, or the presence of tumors in the heart wall, or hydatids, may lead to the rupture.

3. In very rare cases, rupture is produced by stenosis of the aorta and dilatation of the heart cavities.

4. Rupture of the papillary muscles and tendons may be produced by fatty degeneration or inflammatory or ulcerative processes.

ATROPHY.

Atrophy of the walls of the heart may be accompanied with no change in the size of its cavities; or with dilatation (the same as passive dilatation); or, more frequently, with diminution in the size of the cavities.

The atrophy involves most frequently all the cavities of the heart, but may be confined to one or more of them.

The muscular tissue appears normal, or brown from the presence of little granules of pigment in the muscular fibres, which are sometimes present in large numbers; or the muscular fibres may undergo fatty degeneration; or there may be an abnormal accumulation of fat beneath the pericardium; or there may be a peculiar gelatinous material beneath the pericardium: this consists of fat which has undergone mucous degeneration. The heart may be so much atrophied as to weigh four ounces.

The causes of atrophy of the heart are:

1. It is a congenital malformation; the heart of an adult then looks like that of an infant.

2. Any chronic and exhausting disease, repeated hæmorrhages, old age, typhus fever, dysentery, etc., may produce atrophy.

3. Chronic pericarditis, with large, serous effusion, or with thickening of the pericardium, producing constriction of the coronary arteries.

4. Stenosis, atheroma, calcification, or thrombosis of the coronary arteries may produce partial or total atrophy.

5. Myocarditis, with fatty or fibrous degeneration.

6. Mitral stenosis may cause atrophy of the left ventricle.

HYPERTROPHY.

All the cavities of the heart may have their walls hypertrophied, or the thickening may involve one or more. While the wall of a ventricle is thickened, its cavity may retain its normal size—*simple hypertrophy;* or be dilated—*eccentric hypertrophy;* or it may be contracted—*concentric hypertrophy.*

Care should always be exercised in judging of this condition, for a firmly contracted heart seems to have a small cavity and thick walls. The existence of such a condition as concentric hypertrophy is denied by some authors. Eccentric hypertrophy is the most common form.

Simple hypertrophy is not common, but may occur in connection with the atrophied kidneys of chronic diffuse nephritis. The muscle tissue in hypertrophied hearts is firmer and denser than normal, and is apt to have a darker color. Fatty degeneration may, however, be associated with it, giving the walls a lighter appearance. It is probable that the increase of tissue in the hypertrophied heart wall is the result of increase both in size and number of the muscle fibres.

Hypertrophy of both ventricles increases both the length and breadth of the heart. Hypertrophy of the left ventricle (alone) increases its length. The apex is then lower and further to the left than usual. Hypertrophy of the right ventricle (alone) increases the breadth of the heart toward the right side; but sometimes the right edge of the heart retains its normal situation, and the apex is displaced to the left. With large hypertrophy of both ventricles, the base of the heart may sink, so that its long axis approaches a horizontal direction.

Hypertrophied hearts may weigh from forty to fifty ounces, or even more.

Hypertrophy of the heart may depend upon a variety of causes:

1. Changes in the valves; either insufficiency or stenosis in the valves leading from a cavity, and insufficiency in valves leading to a cavity, may induce hypertrophy of its walls.

2. Obstruction to the passage of blood through the arterial system, as in atheroma and other diseases of the intima; congenital or acquired stenosis of vessels, pressure of tumors, etc., on vessels; certain forms of chronic diffuse nephritis, especially atrophied kidneys, lead to hypertrophy of the left ventricle, and sometimes secondarily to hypertrophy of the right ventricle.

3. Obstruction to the passage of blood through the pulmonary artery by stenosis or by certain diseases of the lungs, particularly emphysema and chronic phthisis, may lead to hypertrophy of the right ventricle, and, secondarily, of the right auricle and left ventricle.

4. Any cause, whether muscular or nervous, which increases the rapidity and force of the heart's contractions, may produce hypertrophy.

5. Dilatation of the ventricles, from any cause, is frequently followed by hypertrophy.

6. Pericarditis may produce hypertrophy by inducing softening and dilatation of the ventricles, or by leaving adhesions which obstruct the heart's action. Chronic myocarditis also may lead to hypertrophy.

Finally, for some cases of hypertrophy no satisfactory cause can be found.

It should be borne in mind that an increase in the amount of fat in and about the heart may make the organ appear larger, when there may be actually a considerable decrease in the amount of muscle tissue.

DILATATION.

Dilatation may be combined with hypertrophy—*active dilatation;* or there may be no increase of muscle tissue, but a thinning of the walls proportionate to the dilatation of the cavity—*passive dilatation.*

Either one or all of the heart cavities may be dilated, the auricles most frequently; next the right ventricle; least often the left ventricle.

Active dilatation has been considered under hypertrophy.

Passive dilatation may be produced by:

1. Changes in the valves. Mitral or aortic stenosis or insufficiency may produce dilatation of the auricles and right ventricle. Pulmonary stenosis or insufficiency may produce dilatation of the right auricle and right ventricle. Aortic insufficiency, with or without stenosis or mitral insufficiency, may produce dilatation of the left ventricle. Dilatations from these causes are often succeeded and compensated for by hypertrophy of the heart's walls.

2. Changes in the muscular tissue of the heart walls. Serous infiltration from pericarditis, myocarditis, fatty degeneration and infiltration, atrophy of the muscle fibres, may all lead to dilatation.

3. A heart which is already hypertrophied may, from degeneration of the muscle, become dilated.

4. Acute exudative inflammations of the lungs and acute pleuritic exudations, by rendering a large number of vessels suddenly impermeable to the blood current, may produce sudden stasis in the pulmonary artery and dilatation of the right heart.

5. There are curious cases of acute and chronic dilatation of the ventricles for which no mechanical cause can be found and which are very fatal.

DEGENERATIONS.

Parenchymatous Degeneration of the Heart Muscle.—This lesion frequently occurs in typhoid and typhus fever, pyæmia, erysipelas, and other infectious diseases, as well as in the exanthemata, as a result of burns, and under a variety of other conditions. It is characterized by the presence in the muscle fibres of the heart of greater or less numbers of albuminous granules of various sizes, most of them very small. They are not as refractile as fat-droplets, and are insoluble in ether, while swelling up and becoming almost invisible under the influence of acetic acid. Sometimes they are so abundant as to conceal the striations of the fibres. The degeneration is usually quite uniformly diffused through the heart, whose walls are softer than normal and of a grayish color. This lesion may be associated with or followed by fatty degeneration.

Fatty Degeneration of the Heart Muscle.—This consists in the transformation of portions of the muscle fibres of the heart into fat, which collects in the fibres in larger and smaller droplets, sometimes few in number, sometimes so abundant as to entirely destroy or conceal the normal striations (Fig. 141). These droplets are soluble in ether, and remain unchanged on treatment with acetic acid. This degeneration is sometimes quite universal, but is more apt to occur in patches, giving the heart muscle a mottled appearance. This mottling may usually be best seen on the papillary muscles. The degenerated areas have a pale-yellowish color, and the muscle tissue is soft and flabby; but when moderate or slight in degree, the gross appearance may be little changed, and the microscopical examination be necessary for its determination. This degeneration may lead to thinning of the walls, or to rupture of the heart, or to inability to fulfil its functions. It is not infrequently the cause of sudden death.

It may be secondary to hypertrophy of the heart, to inflammation of the heart muscle, or to pericarditis; to disturbances of the circulation in the coronary arteries by inflammation, atheroma, etc. It may be due to deteriorated conditions of the blood in wasting diseases, excessive hæmorrhages, exhausting fevers, leukæmia, etc., or to poisoning with phosphorus and arsenic. It may occur in otherwise apparently healthy persons.

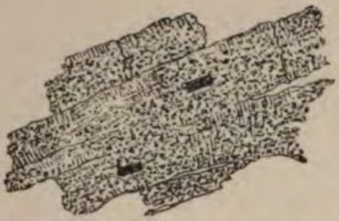

FIG. 141.—FATTY DEGENERATION OF THE HEART MUSCLE. Teased.

Fatty Degeneration of the Endocardium.—It is not uncommon to find, especially in elderly persons, fatty degeneration occurring in patches, especially on the valves, but also on the general endocardium. They may also occur in ill-nourished and anæmic individuals. Small, or even considerable, areas of fatty degeneration appear, as a rule, to be of little or no clinical significance. They are at least not inconsistent with perfect health. In these areas of fatty degeneration, the connective-tissue cells are more or less completely filled with larger and smaller fat-droplets.

Amyloid Degeneration of the endocardium or the walls of the blood-vessels and intermuscular connective-tissue septa is a not very infrequent, but usually not very important lesion.

Calcification of the products of inflammation in pericarditis, or of connective-tissue membranes in chronic pericarditis, sometimes occurs, and in the latter case the heart may be more or less inclosed by a calcareous shell. The muscle fibres of the heart wall may, though rarely, become densely infiltrated with salts of lime.

Fatty Infiltration or Lipomatosis of the Heart.—This lesion, which should be clearly distinguished from fatty degeneration, consists of an unusual accumulation of fat about the heart and between its muscle fibres (Fig. 142).

The subpericardial fat, which may be present in considerable quantity under normal conditions, may be so greatly increased in amount as to form a thick envelope inclosing nearly the entire organ. Sometimes the accumulation of fat extends into the walls of the heart, between the muscles, causing atrophy of the latter, frequently to a very great extent, so that the function of the heart is seriously interfered with. This occurs sometimes in general obesity, or as a result of chronic pericarditis, or in drunkards, or in debilitated or old persons.

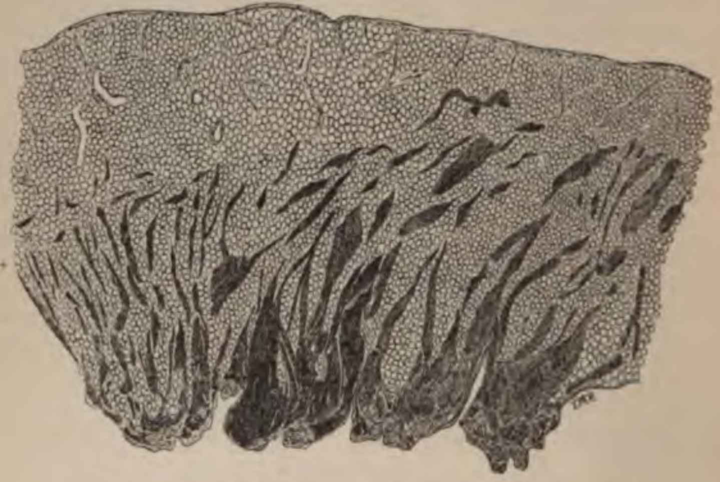

Fig. 142.—Fatty Infiltration or Lipomatosis of the Heart.

The lesion is excessive, the heart muscle being to a large extent atrophied. (The fat-cells are represented in the drawing, for the sake of clearness, of relatively too large size.)

Atrophy of the pericardial fat tissue not infrequently occurs in persons emaciated by chronic disease, and then the usual situations of the fat are occupied by a tissue resembling mucous tissue in its gross characters. Microscopical examination shows that in this atrophic fat the fat-cells have largely lost their contents, and the whole tissue has undergone a partial reversion to its original embryonic form (see Fig. 143).

Myomalacia.—When, through obliterating endarteritis, atheroma, thrombosis, or embolus of a branch of the coronary arteries, the blood-supply is cut off from a circumscribed portion of the heart wall, the tissue in the affected area may undergo fatty degeneration, leading to rupture. Or, instead of extensive fatty degeneration, the muscle fibres may break down into a granular detritus, and the connective tissue about them suffer retrograde metamorphosis, so that the whole affected area may be soft and yellowish-white or grayish in color. If, as not infrequently occurs, there is considerable extravasation of blood, the degenerated area may be of a dark red color. Under these conditions the wall may rupture; or acute inflammatory processes may occur; or the degenerated tissue may be gradually absorbed, and replaced by new connective tissue which gradually grows dense, shrinks, and assumes the characters of cicatricial tissue. This may occur in any part of the heart wall or in the papillary muscles. When the heart wall is involved, the new-formed connective tissue may yield to the blood-pressure from within, and an aneurism of the heart be formed.

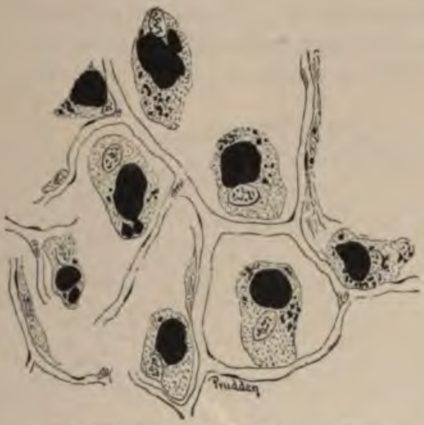

FIG. 143.—ATROPHIC PERICARDIAL FAT.

From young person dead of carcinoma of the stomach and peritoneum. Stained with osmic acid and teased.

INFLAMMATION.

Endocarditis.

The endocardium is a connective-tissue membrane which lines the cavities of the heart and forms its valves. Its inner surface is covered with a layer of endothelial cells. It is but poorly supplied with vessels, and the inflammations which attack it are of the cellular variety. The ordinary products of inflammation, pus, fibrin, and serum, are scanty or absent altogether. The connective-tissue cells and basement substance are principally concerned in the inflammatory processes. The new tissue thus produced is prone to degeneration and calcification The roughening of the endocardium due to the inflammation often causes a coagulation of fibrin on the inflamed surface.

In fœtal life it is the endocardium of the right heart, in extra-uterine life that of the left heart, which is usually inflamed.

The endocardium which forms the valves is that which is most frequently inflamed, but the other portions of it are by no means exempt.

1. *Simple Acute Endocarditis.*—This is most apt to occur in connection with rheumatism, but may occur under other conditions. It may attack a heart which was previously healthy, or one in which the lesions of chronic endocarditis already exist.

In some cases the only lesion is a simple swelling of the valves. They are thick and succulent, but their surfaces remain smooth. The basement substance is swollen, and there is a moderate production of new connective-tissue cells.

In other cases, the growth of connective-tissue cells is very much more marked, the basement substance is split up, and little cellular, fungous masses, called vegetations, project from the free surface of the endocardium. On these roughened surfaces the fibrin of the blood is deposited, and so vegetations of considerable size may be formed (see Fig. 144).

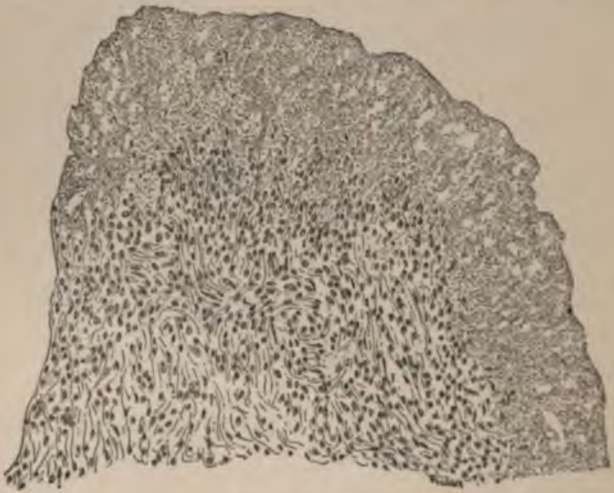

FIG. 144.—VEGETATION ON AORTIC VALVE IN ENDOCARDITIS.
Showing granular thrombus over the surface.

In still other cases the cell growth, while in some places it forms vegetations, in other places degenerates, and thus portions of the valves are destroyed. This is *simple acute ulcerative endocarditis.*

In some cases of this disease, the patients recover, and the valves seem to return to a normal condition; in other cases the valves are left permanently damaged; and in still others chronic endocarditis follows the acute form.

2. *Mycotic or Malignant Endocarditis* (malignant ulcerative endocarditis).

The direct inciting cause of simple acute endocarditis of the forms described above is unknown. But in a considerable number of cases of acute endocarditis, bacteria have been found in and about the vegetations (see Fig. 145), and proved, by careful experiments, to stand in a causative relation to the lesion.

Those cases of acute endocarditis in which the lesions are induced by the direct action of bacteria are called *mycotic* or *malignant* or *bacteritic endocarditis;* or, since the new-formed as well as the old tissue about the bacteria is apt to become necrotic, and thus lead to larger or smaller losses of substance, the lesion is often called *ulcerative endocarditis.*

Cultivations of the bacteria occurring in the heart lesions in malignant endocarditis have shown that, while various species of bacteria may occasionally act as an inciting cause, it is most commonly induced by the Staphylococcus pyogenes aureus and the Streptococcus pyogenes.[1]

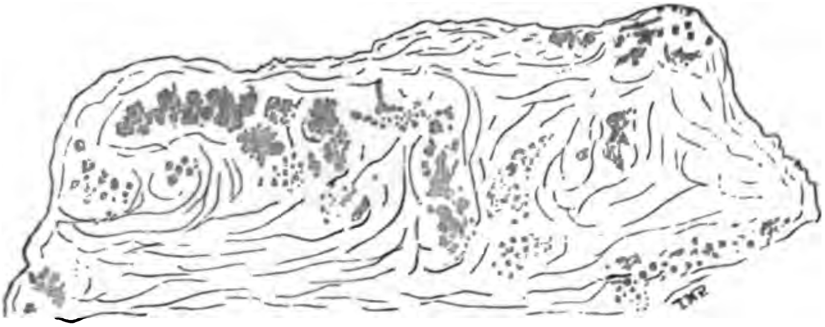

FIG. 145.—MYCOTIC ENDOCARDITIS.
Section of vegetation showing colonies of micrococci, stained with fuchsin.

It has been, furthermore, found that a lesion or injury of the endocardium, either on the heart valves or elsewhere, predisposes to the lodgment and growth upon them of the disease-producing bacteria when once they have gained access to the circulating blood.

Mycotic endocarditis is frequently a secondary complicating lesion, but may occur as a primary disease. It is most apt to be associated with the acute infectious diseases, and in many cases may be regarded as one of the local manifestations of pyæmia

In some cases, there is a formation of new tissue in the form of organized vegetations on the valves or general endocardium; in other cases, necrosis either of the new-formed or the old tissue is the most marked feature. Blood-clots are apt to form on the affected surfaces and often largely make up the so-called vegetations. The mitral and aortic valves are frequently the seat of the lesion, but it may occur elsewhere.

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[1] For a detailed consideration of the relationship of bacteria to malignant endocarditis, with experiments and literature, see *Prudden*, Am. Jour. Med. Sciences, January, 1887.

Detachment of bacteria containing fragments of the vegetations or clots may give rise to single or multiple infectious emboli (see p. 58) and abscesses in various parts of the body, such as spleen, kidneys, brain, skin, heart wall, etc. Bacteria similar to those in the heart lesion may be found in these secondary abscesses (see Fig. 31).

It is probable that these abscesses in ulcerative endocarditis do not always arise from cardiac emboli, but may precede the heart lesion.

3. *Chronic Endocarditis* may succeed acute endocarditis, or the inflammation may be chronic from the outset. It affects most frequently the aortic and mitral valves, and the endocardium of the left auricle and ventricle; similar changes in the right side of the heart being much less frequent.

There are two main anatomical varieties of chronic endocarditis, which may occur separately or together.

(1) The endocardium is thick and dense, its surfaces are smooth or covered with small, hard vegetations or ridges; it is often infiltrated with the salts of lime.

(2) There is a growth of connective-tissue cells in the endocardium, with a splitting-up of the basement substance. Some of the new cells continue to live, others degenerate. By the combination of such a cell growth and destruction, the endocardium is in some places destroyed, in others changed into projecting vegetations. Fibrin is deposited on the roughened surfaces. After a time, the condition may be further complicated by the shrinkage and deposition of the salts of lime in the new tissue and in the endocardium. All these changes may extend to the wall of the heart beneath the endocardium.

The most important result of chronic endocarditis is its effect on the heart valves, producing insufficiency and stenosis. The changes in the valves are followed by changes in the walls and cavities of the heart, and disturbances of the circulation throughout the body.

4. *Chronic Ulcerative Endocarditis.*—Large ulcers or perforations of the valves may be formed in chronic endocarditis, upon which clots may form, so that in gross appearance a great similarity exists between this and malignant ulcerative endocarditis, particularly if the latter have been ingrafted upon an already chronically diseased endocardium. The microscopical and biological examinations must usually be resorted to in order to determine the exact significance of the lesion.

5. *Tubercular Endocarditis* may occur in connection with tubercular pericarditis or general miliary tuberculosis. The tubercles may be

small and single, or grouped in masses, and show the usual degenerative changes.

Myocarditis.

The inflammatory changes in the walls of the heart involve primarily the interstitial tissue and blood-vessels, the muscle fibres being secondarily affected by atrophic and degenerative changes.

There is a change in the muscle fibres by which they are broken into rectangular fragments, with a collection of granular matter around the nuclei, the entire tissue becoming dense and translucent. This change is sometimes called "parenchymatous myocarditis," but its inflammatory nature is doubtful.

Interstitial Myocarditis may be acute and purulent, or chronic with the formation of new connective tissue.

Acute Purulent Myocarditis may be diffuse, infiltrating the wall of the heart with pus. This may occur as a complication of scarlatina and from unknown causes.

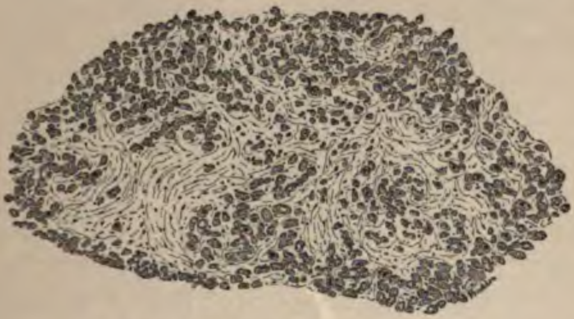

FIG. 146.—CHRONIC INTERSTITIAL MYOCARDITIS.
Showing transverse section of a portion of a papillary muscle.

More frequently the purulent inflammation is circumscribed, producing abscesses. These occur with pyæmia, mycotic ulcerative endocarditis, and other infectious diseases. They are of different sizes, and either single or multiple. They are produced by the lodgment of infectious emboli in small vessels. The contents of the abscesses consist of pus, broken-down muscle tissue, and bacteria. These abscesses may open into the pericardial sac and set up a purulent pericarditis; or into a heart cavity, giving rise to thrombi in the heart and emboli in different parts of the body; or the wall of the heart is weakened by the abscess, so that it ruptures, or an aneurismal sac is formed; or an abscess in the interventricular septum may establish an opening between the ventricles; or the suppurative process may extend upward and form an abscess in the connective tissue at the base of the heart.

In rare cases, the patients recover, the contents of the abscesses become dry and hard, and inclosed by a wall of fibrous tissue.

Chronic Interstitial Myocarditis may be secondary to pericarditis or endocarditis, to obliterating endarteritis of the branches of the coronary artery, myomalacia, or may occur by itself. There is a growth of new connective tissue or of granulation tissue between the muscular fibres, with atrophy and degeneration of the muscle. This growth may be in the form of circumscribed patches (Fig. 146), or diffused over a considerable part of the wall of the heart. Such an interstitial inflammation is often followed by dilatation of the cavities of the heart, by the formation of aneurisms of the wall of the heart, and of thrombi in the cavities of the heart.

Syphilitic Myocarditis is accompanied by the growth of connective tissue or granulation tissue in the wall of the heart between the muscular fibres. The pericardium and endocardium may also be thickened, and pericardial adhesions may be formed. Gummata of the heart are of rare occurrence.

CHANGES IN THE VALVES.

Fenestration of the valves is usually a change productive of no bad consequences. It occurs very frequently in the aortic and pulmonary valves. The valves may be thinner than usual, and close to their free edges are small slits extending from the centre to the attached edges of a leaf.

Aneurisms of the valves are produced in two ways:

1. They are the result of endocarditis. One of the lamellæ of the leaf of a valve is destroyed, and the other lamella is converted into a sac filled with blood. These aneurisms are found in the aortic valve, projecting into the ventricle; and in the mitral valve, projecting into the auricle. Not infrequently the wall of the aneurism gives way, so that there is a rupture entirely through the valve.

2. The entire thickness of a leaf of a valve is converted into a sac filled with blood. This occurs in the aortic, mitral, and tricuspid valves; its cause is unknown.

Hæmorrhage in the substance of the valves is sometimes found in very young children. It does not appear to have much clinical importance.

ANEURISM OF THE HEART.

Sacs filled with blood, situated in the walls of the heart and communicating with its cavities, are formed in several different ways.

1. In consequence of inflammatory processes in the endocardium and muscular tissue, a small or large portion of the wall is converted into fibrous tissue. The portion thus changed no longer resists the pressure

of the blood from within, and is driven outward. Such a pouch may be a circumscribed sac, communicating with the heart cavity by a small opening, or may look like a dilatation of part of the ventricle. The wall of such an aneurism becomes thinner as the sac increases in size. It is composed of the endocardium, new fibrous tissue, visceral pericardium, and sometimes the adherent parietal pericardium. The walls may calcify, or rarely they become so thin as to rupture externally or into the right ventricle. The sacs may contain fluid blood or be filled up with fibrin.

Such aneurisms are usually situated in the wall of the left ventricle; rarely in that of the left auricle. If they are in the septum, they may project into the right ventricle. They are usually single, but sometimes two or three are found in the same heart.

2. Fatty degeneration of the heart wall may reach such a point that the wall yields and is pouched out into an aneurismal sac.

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3. Endocarditis and myocarditis, or fatty degeneration, may so soften a portion of the heart wall that the endocardium and part of the muscular tissue are ruptured, and a ragged cavity is formed. This form of aneurism usually does not attain a large size, but soon ruptures externally, and causes the death of the patient.

THROMBOSIS OF THE HEART.

It is very common to find after death, in the heart cavities, yellow, succulent, semi-translucent masses. They are most common and of firmest texture in persons who die of acute inflammatory diseases. They may adhere quite firmly to the walls of the heart, and may extend in long, branching cords into the vessels. They are formed in the last hours of life and just after death. They have no clinical or pathological importance.

Coagulations of the fibrin of the blood in the heart do, however, occur during life, and may exist for years. If the fibrin adheres to the valves in small masses, these are called vegetations; if it coagulates in the heart cavities in larger bodies, they are called thrombi or heart polypi.

Such thrombi are found in all the heart cavities. They form flattened masses firmly adherent to the endocardium; or rounded bodies in the spaces between the trabeculæ; or have a polypoid shape and are attached by a narrow pedicle, or are free in the cavity.

They are usually found in connection with some valvular lesion which prevents the free circulation of blood through the heart.

They are firm, dry, and of a whitish color; they may soften and break down at their centres, so as to look like cysts filled with pus, or they may calcify. They are usually entirely unorganized, consisting simply of fibrin.

One of us (Delafield) has seen an organized thrombus in the heart of a man, whose history was unknown, who was found dead in the street.

Sometimes sarcomatous and carcinomatous tumors in different parts of the body are accompanied by the formation of thrombi in the heart cavities which are composed partly of coagulated blood, partly of tissue like that of the primary tumor.

TUMORS.

Primary tumors in the heart are rare; but *sarcomata, myxomata, fibromata,* and *lipomata* may occur. *Rhabdomyomata,* probably congenital, may occur in the heart wall as circumscribed nodular masses. A *cavernous* tumor of this kind has been described. Secondary tumors, as a result of metastasis, or of continuous growth from adjacent parts, are not very infrequent. These are usually *carcinomata* or *sarcomata.* Secondary *chondromata* have been observed.

PARASITES.

Echinococcus sometimes occurs in the heart wall and may perforate into the cavities. Cysticercus cellulosæ has been observed.

THE BLOOD-VESSELS.

ATROPHY AND HYPERTROPHY.

Atrophy of the blood-vessels may involve the entire trunk or some of its elements. It may occur as a part of general malnutrition of the body, or in connection with atrophy of particular organs, or as an accompaniment of various diseases of the vessels themselves.

Hypertrophy, which is especially seen in the arteries, may occur in the establishment of a collateral circulation upon the closure of arterial trunks, or it may occur as the result of increased blood-pressure, as in some forms of hypertrophy of the heart.

DEGENERATION.

Fatty Degeneration.—This may occur in the walls of otherwise unaltered vessels, or in those which have undergone a variety of inflammatory or degenerative changes. It may occur either in the intima or media, or both, and may be so extensive as to form a very prominent gross lesion, or so little developed as to require the microscope for its recognition. When marked, especially if occurring in the intima of large vessels, smaller and larger spots or stripes or patches may be seen, of a yellowish-white color, usually sharply circumscribed, and sometimes smooth, sometimes roughened on the surface. It is most apt to occur in the aorta, but may be found in any of the vessels. In moderate degrees of the lesion, we find on section that the cells of the intima contain fat-droplets in greater or less number. When further advanced, not only are the cells crowded with fat-droplets, but the intercellular tissue also may be more or less densely infiltrated with them. Sometimes the infiltration is so dense that the tissue breaks down, and there may be an erosion of the surface, forming a so-called fatty ulcer. When the media is involved, the muscle cells contain fat-droplets. It may lead to the formation of aneurism or to rupture of the vessels.

Calcification usually occurs in vessels otherwise diseased, and may involve either the intima or media. It consists in the deposition of salts of lime either in the cells or intercellular substance. The lime may be in the form of larger or smaller granules or in dense translucent plates.

Amyloid Degeneration, which may affect all the coats of the arteries, but especially the intima and media, will be considered under the lesions of the organs in which it most commonly occurs.

Hyaline Degeneration may cause thickening of the intima of the blood-vessels by its conversion into or infiltration with a homogeneous material somewhat similar to amyloid (see page 68). Or it may involve the entire wall of smaller vessels, converting them into irregular lumpy cords. The lumen of vessels thus changed may be obliterated or occluded by thrombi.

THE ARTERIES.

INFLAMMATION.

Acute Arteritis.

Acute inflammation of the walls of the arteries is, in the majority of cases, the result of injury, or of an inflammation in the vicinity of the vessel, or of the lodgment within it of some foreign body of an irritating or infectious nature. The inflammatory process may be largely confined to the inner layer of the vessels—*endarteritis;* or it may commence in the outer layers—*periarteritis;* or it may involve the entire wall.

The blood-vessels in the outer layers may be congested, the tissue œdematous and infiltrated with pus cells, and the entire wall may become necrotic. The intima, if this layer is involved, loses its natural gloss, looks dull and swollen. It may become infiltrated with pus from the outer layers, and it may become necrotic. Under these conditions thrombi usually form, and in these may occur the various changes which have been already described on page 56.

Chronic Arteritis.

In chronic arteritis, all the coats of the vessel may be involved, but i most cases the lesion is either largely confined to, or most marked in, t intima—*endarteritis.* The disease may supervene upon an acute inflammation of the artery, or it may be chronic from the beginning. The inflammation may occur in patches or in irregular segments of the vessels, of various lengths, or it may occur diffusely. It may be limited to single arterial trunks, or it may affect more or less all the arteries of the body. Arteries which are the seat of slight degrees of chronic inflammation may appear to the naked eye but slightly, or even not at all, changed, or the walls may seem stiffer than usual, and remain widely open when cut across. When the lesion is more marked, whitish patches or areas of distinct thickening of the wall may be seen, which sometimes

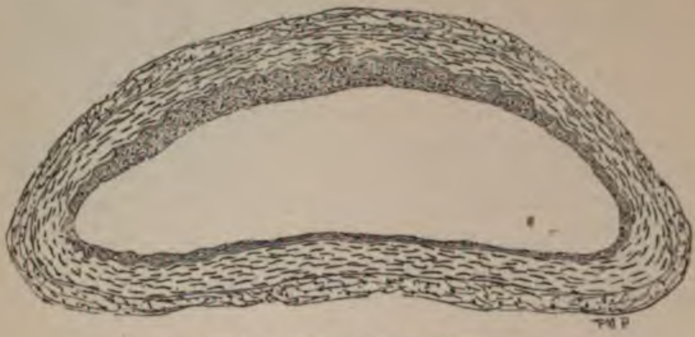

FIG. 147.—CHRONIC ENDARTERITIS.

Involves one of the cerebral arteries. The amount of thickening of the intima is in this case moderate.

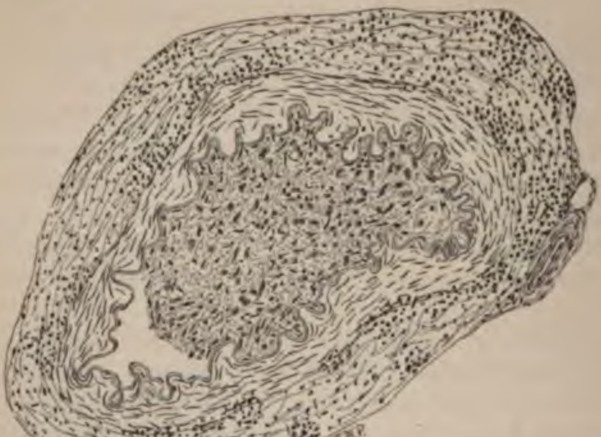

FIG. 148.—CHRONIC OBLITERATING ENDARTERITIS.

The lesion in this case is excessive, almost entirely closing the lumen of the vessel. There is, too, a moderate degree of periarteritis.

visibly incroach upon the lumen of the vessel (Fig. 147). Microscopical examination shows that the thickening of the wall is due to the formation of new connective tissue, mostly in the intima. The new tissue may be soft and gelatinous in character and contain few or many cells; or it may be very dense and hard and contain very few cells. The endothelial layer of the intima may remain intact over the thickened area. Parts of the vessel not distinctly thickened may contain an unusual number of small spheroidal cells. Sometimes the musculosa and adventitia as well as the intima show at the seat of thickening an increase of new connective tissue. The increase of connective tissue in the intima, particularly of smaller arteries, may be so great as to incroach seriously upon the lumina, and even lead to their obliteration—*endarteritis obliterans* (Fig. 148). Obliterating endarteritis is very common in the interstitial inflammation of organs, such as the kidney, liver, etc., as a part of the general new formation of connective tissue. Arteries which are the seat of chronic arteritis are not infrequently the seat of secondary acute inflammatory changes, so that, in addition to the formation of connective tissue, they may become infiltrated with small spheroidal cells, which may lie singly or in clusters in the various layers, and particularly along the blood-vessels of the adventitia.

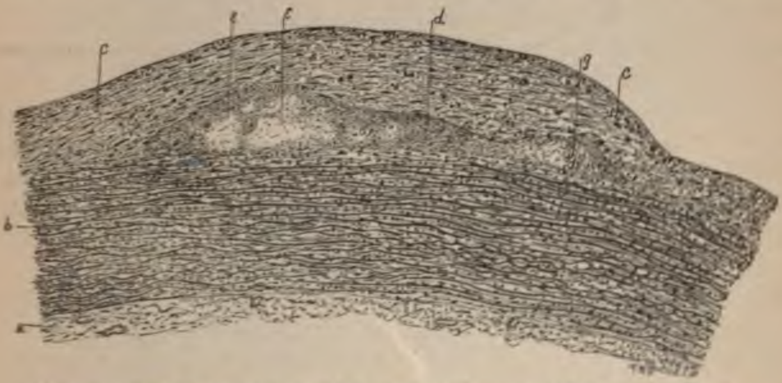

FIG. 149.—ATHEROMA OF THE AORTA, WITH DEGENERATION OF NEW-FORMED TISSUE.

a, adventitia; *b*, media; *c*, new tissue developed in the intima; *d*, degenerated area; *e*, area of softening; *g*, fat-droplets in softened area.

Arteries which are the seat of chronic endarteritis are liable to suffer a series of degenerative changes, consisting chiefly of fatty degeneration, calcification, or the breaking-down of the degenerated tissue, and the formation of inclosed softened areas (Fig. 149), or erosions or ulcer-like openings in the intima. To the varied alterations produced in the walls of the arteries by combinations of these inflammatory and degenerative changes the name *atheroma* is frequently applied.

When fatty degeneration supervenes upon chronic endarteritis, the thickened areas may have a light-yellowish color, and the microscopical examination will show a greater or less amount of fat in and between the cells of the intima or the new tissue produced therein. Should the fatty degeneration be extensive, the basement substance and the cells may gradually disintegrate, and larger and smaller irregular cavities may be formed in the walls, filled with fat-droplets and tissue detritus.

These softened areas, sometimes called *atheromatous cysts*, frequently also contain cholestearin, and there may be partial calcification of their contents, or of the tissue of the arterial wall about them. Sometimes the wall of the artery, instead of undergoing simple fatty degeneration, or in connection with this change, becomes in circumscribed localities looser in texture by the conversion of the muscular and connective tissue into a finely fibrillar substance which may persist for some time or break down, forming an atheromatous cyst. These atheromatous cysts may, through a degeneration of the tissue inclosing them, open into the lumen of the vessel, forming the so-called *atheromatous ulcers*. Fragments of tissue from the edges of these ulcers, or from the contents of the cysts, may be carried into the circulation, forming emboli. Fibrinous thrombi are prone to form upon the roughened surface of the intima or upon the surface of the erosions. Very frequently the calcification is so extensive that large, hard, calcareous plates are formed in the walls and may project inward, forming roughened surfaces on which thrombi are deposited. Fatty degeneration and calcification of the media and thickening of the adventitia may occur in connection with atheroma of the intima. Sometimes the vessels by these changes become greatly deformed, so that over considerable areas the entire wall may be altered, presenting raised and roughened surfaces interspersed with calcareous plates, erosions, thrombi, etc. Chronic endarteritis and atheroma is most common and marked in the aorta, but it may occur in smaller arteries, particularly in the cerebral and coronary arteries.

Arteritis as a result of *syphilis* is of frequent occurrence. This may lead to partial or complete obliteration of the lumen of the vessel by a thickening of the intima (Fig. 95), or it may involve all the arterial coats. Histologically the new tissue formed in syphilitic arteritis is identical in most cases with the product of simple chronic inflammation; but when arteries are involved in the growth of gummy tumors, the cheesy degeneration characteristic of the latter often affects the vessels also. Syphilitic arteritis is apt to affect smaller vessels, and the degenerative changes do not form so prominent a feature as in other forms.

In tubercular inflammation, the walls of the arteries, particularly the smaller ones, may be thickened, and their lumina obliterated (Fig. 150).

The results of arteritis vary greatly, depending upon the size and situation of the affected vessel, and the extent and particular form which the lesion assumes. The vessels may become dilated by a yielding to the blood-pressure of their weakened walls, and aneurisms of various kinds may be thus produced. Thrombi and emboli may be induced by means of the roughened surfaces common in endarteritis, particularly of the larger vessels. On the other hand, obliterating endarteritis may cause a partial or entire shutting-off of the blood-supply to a part, and a more or less serious interference with its nutrition, or even its death. Hypertrophy of the left ventricle of the heart may accompany extensive arteritis, as a result of the increased rigidity of the walls of the vessels, or the diminution of their lumina.

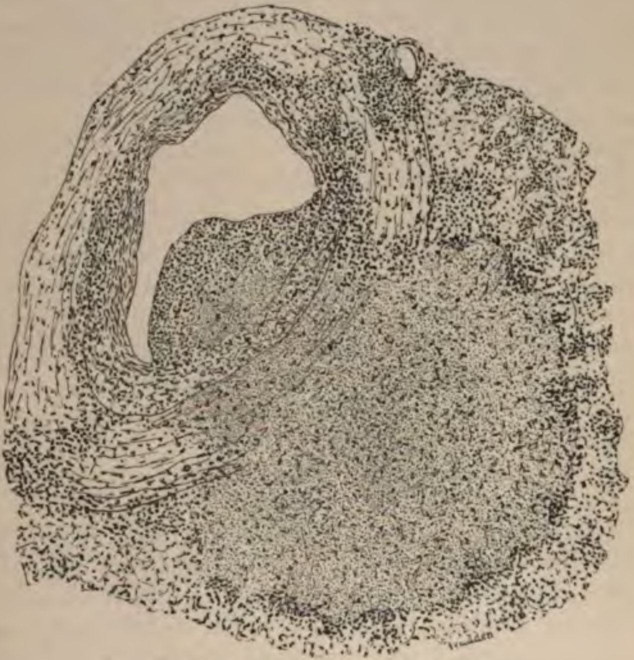

FIG. 150.—TUBERCULAR ARTERITIS IN THE LUNG.

Showing the incroachment of an area of tubercular inflammation upon the wall of the artery and the formation of a mass partly occluding the lumen of the vessel. This section shows how the generalization of the tubercular inflammation through the body may occur by the sweeping away of the tubercle bacilli by the blood and the establishment of new foci in various parts of the body. From specimen prepared by Dr. J. S. Ely.

It is important to remember that a very slight contraction of arteries which have undergone a considerable degree of obliterating endarteritis may produce, for the time being, the same effect upon the part supplied by them with blood that a permanent occlusion would do. Such an occlusion of the vessels and cutting-off of the blood-supply may be of the greatest significance, even though it be but temporary, if it involve branches of the coronary or cerebral arteries. Although it is not yet proven that temporary contraction of partially occluded arteries is a sufficient or frequent cause of sudden death, there are cases of sudden death with the symptoms of heart failure or with the symptoms of hemiplegia, in which the only discoverable lesions are obliterating endarteritis, respectively, of the coronary or cerebral arteries. We have seen cases of sudden death occurring with symptoms of hemiplegia, in which there was, at some point of the middle cerebral artery, considerable, although not complete, obliteration of the vessel, but no evidence of degeneration of the brain tissue supplied by it.[1]

The causes of chronic arteritis and atheroma are in many cases not understood. It may commence in an acute process, or it may be slow in its development. It is very common in old people, and seems often to be of no particular significance. On the other hand, it may, as we have seen, particularly in the smaller arteries, be associated with syphilis, tuberculosis, or interstitial inflammation of organs.

DILATATION AND ANEURISM.

1. *Cirsoid aneurism* consists in the dilatation and lengthening of large or small arteries. The walls of the artery are thinned, the vessel is tortuous and in places sacculated. These changes are most frequent in small arteries, especially the temporal and occipital; they involve the trunk of the vessel and its branches, or may extend to the capillaries and small veins. They form larger or smaller tumors beneath the skin.

Rarely they are found in the larger arteries, and even in the aorta.

2. *The ordinary aneurism* is a dilatation of the coats of the artery over a larger or smaller part of its course. Such dilatations are usually due to chronic endarteritis and atheroma. According to their shape, we may distinguish two varieties: the diffuse and the circumscribed.

(*a*) The diffuse, cylindrical, or fusiform aneurism consists in a uniform dilatation of all the coats of an artery, so that it assumes the shape of a fusiform or cylindrical swelling. In the walls of the dilated portion of the vessel there are often smaller, circumscribed dilatations. The wall of the aneurism is atheromatous or calcified; the middle coat may be atrophied. The arch of the aorta is the most common seat of this form of aneurism, but the entire length of the aorta, or parts of any other arteries, may be dilated in the same way.

(*b*) The circumscribed or sacculated aneurism consists either in a dilatation of the entire circumference of an artery over a short portion of its length, or in a dilatation of only a small portion of one side of the wall, so that the aneurism looks like a swelling attached to one side of the artery. The aneurism commences as a dilatation of all the coats of the vessel; but as soon as it attains any considerable size, the middle coat atrophies, so that the wall is composed of the inner and outer coats; or the inner coat is destroyed by endarteritis, so that the outer coat alone forms the wall of the aneurism. As the aneurism increases in size, it presses upon and causes the destruction of the neighboring tissues and viscera, and portions of these tissues and viscera become incorporated with or take the place of the wall of the aneurism. The cavity of the aneurism is filled with fluid or clotted blood, or with layers of fibrin which adhere closely to its wall. The communication between the aneurism and the artery may be small or large. If arterial branches are given off from the aneurism, they may remain open or become plugged with fibrin; or their walls are thickened and their cavities narrowed by endarteritis. Death is produced by the pressure and interference of the aneurism with the adjoining viscera, or by rupture. The rupture may allow enough blood to escape to destroy life, or the blood may be held in by the soft parts, and a second false aneurism formed about the original one.

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[1] Consult *Cohnheim* and *v. Schulthess-Rechberg*, "Ueber die Folgen d. Kranzarterienverschliessung für das Herz," Virch. Archiv, Bd. 85, p. 503; also *Leyden*, "Ueber d. Sclerose der Coronar-Arterien u. d. davon abhängigen Krankheitszustände," Zeitsch. f. klin. Med., Bd. vii., pp. 459 and 539.

Dissecting aneurisms are those in which, owing to a solution of continuity of the inner layers of the artery, the blood gets between the media and adventitia, and forces its way for a greater or less distance between them. Or it may separate the media into two layers.

ANEURISMS OF THE DIFFERENT ARTERIES.

The aorta may be dilated over its entire length; or there may be diffuse or circumscribed dilatations at any portion of its course; or there may be several aneurisms, situated at different points. The ascending portion of the arch of the aorta may be uniformly dilated in a fusiform shape; or there may be circumscribed dilatations on its anterior wall, or, more rarely, on its posterior wall. The sacculated aneurisms may be of all sizes, and may rupture within the pericardium; or they may form a cavity in the upper part of the ventricular septum, and communicate by openings into the pulmonary artery and left ventricle; or they may dilate downward between the visceral and parietal pericardium, in front of the heart, pushing that organ backward. They may perforate into the right or left auricle or right ventricle, the superior vena cava, or the pulmonary artery; or they may reach a large size, press on and erode the right side of the sternum and adjoining ribs, project under the skin, and even rupture externally.

The transverse portion of the arch may be dilated in a fusiform shape; or there may be sacculated aneurisms at any point in its wall. The sacculated aneurisms usually reach a considerable size. They press on the sternum and ribs in front, or on the œsophagus, trachea, and bronchi behind. The large arteries given off from the arch may be occluded. They cause death by pressure on the air passages, the œsophagus, and the vena cava; or may rupture externally or into the œsophagus, trachea, bronchi, or pleural cavities.

On the abdominal aorta we usually find aneurisms sacculated. If they are situated high up, they may project into the pleural cavities; if lower down, into the abdomen. They may compress and displace the viscera, vessels, and nerves, and erode the vertebræ. They may rupture behind the peritoneum, into the peritoneal cavity, the pleural cavities, the inferior vena cava, the lungs, the colon, the pelves of the kidney, or the posterior mediastinum.

The coronary arteries may be dilated throughout, or may be the seat of small sacculated aneurisms. These may rupture into the pericardium, or may cause rupture of the heart wall.

The pulmonary arteries are rarely the seat of aneurisms. Diffuse and circumscribed dilatations, however, sometimes occur on the main trunk and on the two principal branches of the artery. They do not usually reach a large size, but may cause death by rupture. General dilatation of all the branches of the pulmonary artery is more common. It is found in connection with stenosis of the mitral valves, and with compression or induration of the lung tissue.

Of the other arteries of the body, there is hardly any one which may not become the seat of an aneurism, but those of the popliteal artery are most common.

STENOSIS.

Stenosis and obliteration of the aorta, at the point of entrance of the ductus arteriosus, have been described in a considerable number of cases.

The situation of the stenosis is either exactly at the entrance of the ductus arteriosus or close on either side of this point. The degree of stenosis varies. The aorta may be entirely closed and converted into a solid cord for a length of half an inch; or there may be a circular constriction through which there is a larger or smaller opening—the constriction is uniformly circular; or there is a septum springing from the concave side of the vessel at the opening of the ductus arteriosus; or there is a cicatricial-like contraction of the aorta. The walls of the aorta at this point may be thickened and sclerosed. The ductus arteriosus may be closed or open. Above the constriction the aorta is usually dilated; below it, it is normal, dilated, or stenosed.

Stenosis of the aorta produces hypertrophy of the left ventricle, and, later, of the right ventricle, with venous congestion throughout the body; or there may be a collateral circulation developed between the arteries given off above and below the constriction; or there may be rupture of the aorta, the right ventricle or auricle.

This condition is found at all ages, but is produced during fœtal life or in the first year of extra-uterine life. It is probable that it may be caused after birth by an abnormal closure of the ductus arteriosus. This vessel normally becomes closed without the formation of a thrombus. If a thrombus is formed, it may extend into the aorta and obstruct it; or the ductus arteriosus is filled with a thrombus, but increases for a time in size; afterward, as the thrombus is absorbed, the vessel contracts and draws the walls of the aorta together.

Stenosis of the aorta and of some of the other arteries has been observed, in a few rare cases, without any known cause.

Endarteritis, with the production of atheromatous and calcareous patches, may obstruct or entirely obliterate the smaller arteries. This is especially seen in the arteries of the leg, foot, and brain, and in the coronary arteries. The writer (T. M. P.) has seen a case in which the subclavian was completely occluded in this way.

Narrowing of the aorta and of all its branches, with thinning of the arterial coats, is found as a congenital condition. It usually occurs in females, in connection with imperfect development of the whole body.

Stenosis from thrombosis or embolism is treated of elsewhere.

RUPTURES AND WOUNDS.

Rupture of arteries may occur under the following conditions:

1. Fatty degeneration or endarteritis, with atheromatous changes, may so soften and destroy the inner and middle coats of an artery as to admit of its rupture. The aorta, just above the valves, is the most frequent seat of this lesion. The rupture may run in any direction; its edges are irregular and jagged. The blood may burst through all the coats of the aorta at the same point; or, more frequently, the external coat remains and the blood is infiltrated in the middle coat and between it and the external coat. In this way, a *dissecting aneurism* is formed, which may extend along the aorta for a considerable distance. After a short time, the external coat usually gives way at some point, and the blood escapes. In rare cases, life is prolonged for some time, the rupture being closed by a new membrane.

We also find ruptures from fatty degeneration and atheroma in the arteries of the brain and lungs; in the coronary arteries, the cœliac axis, the mesenteric arteries, and in the arteries of the extremities.

2. In rare cases, stenosis of a portion of the aorta may cause rupture at some point between the seat of stenosis and the heart.

3. Contusions, wrenchings, and severe falls may rupture the walls of an artery, either partially or completely, producing traumatic or dissecting aneurisms, or completely severing the vessel.

4. Penetrating wounds may injure or entirely sever an artery. If the vessel be large and the injury severe, death from hæmorrhage is

the usual result. A small artery may become closed or be the seat of a false aneurism.

In the healing of a wounded artery, two conditions co-operate. The vessel retracts and contracts, and a thrombus is formed within it. The contraction may be alone sufficient to close the vessel; its coats thicken, and the inner surfaces finally are fused together; or the blood coagulates, and forms a thrombus in the vessel near the wound. This thrombus later becomes organized, and the vessel is converted into a fibrous cord.

Spurious or false aneurisms are found most frequently connected with vessels of the extremities. When an artery is wounded, the blood escapes into the surrounding soft parts, and a cavity is formed filled with blood and broken-down tissue. This condition may terminate in several ways.

(*a*) The wound in the artery may heal and the effused blood be absorbed.

(*b*) The effused blood and broken tissues may become gangrenous and the surrounding soft parts be inflamed.

(*c*) A sort of sac wall may be formed by the soft parts, while the wound of the artery remains open, so that we have an aneurismal sac through which the blood is constantly pouring.

5. If an artery be wounded, and at the same time the vein which accompanies it, we have as the result the conditions called aneurismal varix and varicose aneurism. In aneurismal varix, the artery and vein become adherent at the seat of injury, so that the arterial blood passes directly into the vein. There is a smooth, rounded opening between the two vessels, the vein is dilated into a sac, and the veins emptying into it are dilated and tortuous.

In varicose aneurism, the artery and vein do not communicate directly, but a false aneurismal sac is formed between the vessels, into which the blood is poured before passing into the vein.

Varicose aneurism may also be produced by the spontaneous rupture of an aneurism into a vein. The aneurism presses against the vein, becomes adherent, and finally ruptures into it. This condition has been observed between the aorta and pulmonary artery; the aorta and inferior and superior vena cava; the popliteal artery and vein; the femoral artery and vein; the splenic artery and vena azygos; the internal carotid and sinus cavernosus. Even in cases of perforation by aortic aneurisms, life is usually prolonged for some time.

6. Destructive inflammation or tumors of the surrounding tissues may invade and destroy a portion of the wall of an artery. Thus ulceration of the trachea, bronchi, bronchial glands, and œsophagus, or tumors of these parts, may perforate the aorta; gangrene of the lungs, the pulmonary arteries; ulcer of the stomach, the gastric arteries, etc.

Tumors.—Secondary tumors, chiefly carcinomata and sarcomata, may occur in the walls of the arteries by continuous growth from without, involving first the external layers. To these layers they are usually confined, for the density of the inner layers affords such marked resistance to the infiltration of the tumor cells that they are apt to pass intact through the tumor which grows around them. More frequently the arteries become secondarily involved in the growth of malignant tumors by the occurrence, within them, of emboli formed by larger and smaller masses of tumor cells.

These emboli are usually of small size, and are apt to get into the circulation by growing through the walls of the veins into their lumina. Large emboli from tumors are most apt to occur in the branches of the pulmonary artery. The emboli, formed as they are for the most part by cells capable of growth and proliferation, are apt to soon form connection with the walls of the vessels, and by the growth into them of blood-vessels from the *vasa vasorum* to find the conditions necessary for their development, and they may thus soon involve the entire wall of the vessel and grow out into adjacent parts.

THE VEINS.

DILATATION.

Dilatation of the veins, or phlebectasia, presents itself under a variety of forms.

1. *Simple Dilatation.*—The vein is uniformly dilated in a cylindrical or fusiform shape; its length is not increased; its walls are of normal thickness or thinned; the valves increase in size, or are insufficient, or atrophy, or are torn.

2. *Cirsoid Dilatation.*—The vein is uniformly cylindrically dilated, but is also increased in length, so that it assumes a very tortuous course. The walls are normal, thickened, or thinned.

3. *Varicose Dilatation.*—A circumscribed portion of the wall of the vein is dilated so as to form a globular sac. The sac communicates with the vein through a large or small opening. The wall of the sac is formed of the coats of the vein, which preserve their normal thickness, are thickened or thinned; the middle coat may disappear entirely. There may be only one such dilatation, or there may be a number on the same vein, or a number of veins may be affected at the same time. The vein may be otherwise normal, or, more frequently, is dilated in the cirsoid form.

4. *Anastomosing Dilatation.*—A number of contiguous and anastomosing veins are dilated, both in the cirsoid and varicose forms. The vein then looks like a series of cavities separated by thin partitions. The dilatations of the same vein become adherent to each other and to those of the adjoining veins; portions of the wall of the dilated parts may dis-

appear, and we find a number of cavities containing venous blood, and separated from each other by thin partitions. The course of the vein can no longer be followed out.

Spontaneous cure of dilatations of the veins is not common, and usually occurs only in the lesser degrees of the lesion. Most phlebectasiæ increase steadily in size and extent. Very frequently thrombi form in the dilated veins, and either partially or completely fill them; and these in rare cases may become organized, or the clots may dry and become calcified, forming *phleboliths* (see page 56), and, by the formation of new connective tissue in the walls, they may become inclosed in a fibrous capsule, with the obliteration of the vessel. The wall of the dilated sac may become so thin that it finally ruptures, and the blood is discharged externally. Sometimes inflammation is set up in the tissues surrounding the vein, and we find both the surrounding tissues and the wall of the vein the seat of purulent infiltration or fibrous thickening. The parts of the body from which the dilated veins draw their blood may exhibit the results of chronic venous congestion, œdema, hyperæmia, and hypertrophy or ulceration.

When occurring in mucous membrane, dilated veins are usually associated with persistent catarrh. There is hardly one of all the veins of the body which may not be dilated. The hæmorrhoidal veins, the veins of the leg and thigh; those of the pelvis and pelvic viscera; those of the spermatic cord, scrotum, and labia; those of the abdominal wall; those of the neck and arms—are the ones most frequently found in this condition.

The causes of dilatation are principally some mechanical obstruction to the passage of the blood through the veins towards the heart; but changes in the walls of the vessels from inflammation or injury, etc., are not without influence.

WOUNDS—RUPTURE.

Wounds of the veins usually heal by a simple contraction and an adhesive inflammation of their walls: sometimes by the formation of a thrombus. *Rupture* of the veins may be produced by severe contusions and crushings of the body, and by violent falls. *Perforation* of a vein may be produced by suppuration of the soft parts, and the invasion of the walls of the vessel; by the pressure of an aneurism or of a new growth; by the thinning of the wall of the vein in phlebectasia.

INFLAMMATION.

Inflammation of the veins, *phlebitis*, may involve chiefly the external layers—*periphlebitis;* or the internal—*endophlebitis;* or, as is very frequently the case, the entire wall may be affected. Phlebitis may be caused by the presence of a thrombus, by injuries, or by an inflammation of the surrounding tissues. Thrombosis of the vein, either primary or secondary, is a very constant accompaniment of phlebitis.

Acute Phlebitis may commence as a suppurative periphlebitis or as a result of inflammatory processes about the vessel. The outer layers of the venous wall are congested, swollen, infiltrated with serum and pus. The inner coats may become infiltrated with pus: they may become necrotic and disintegrate. A thrombus is constantly formed under these conditions, which may for a time stop the circulation and keep the products of inflammation and degeneration from mixing with the blood; but the thrombus itself is prone to disintegration, and thus the exudations and decomposing fragments of tissue may enter the circulation.

On the other hand, owing to the presence of irritating or infectious material within the vein and the formation of a thrombus, the inflammatory process may be at the commencement an endophlebitis, but usually, if the inflammation be at all severe, the entire wall of the vessel will eventually be involved. The pus cells in both cases doubtless come from emigration from the vasa vasorum. Acute phlebitis may terminate in the absorption of the thrombus and the return of the vein to its normal condition; in the obliteration of the vein; or portions of the thrombus may become detached, and find their way as emboli into various parts of the body. The most important results of phlebitis are usually those which depend upon the introduction into the blood of these emboli or of septic material (see Thrombosis and Embolism, page 55, and Pyæmia).

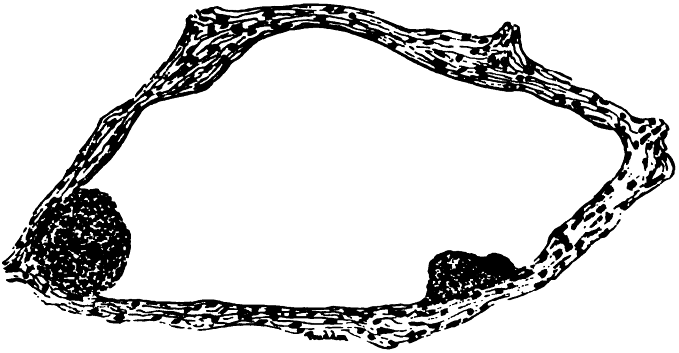

FIG. 151.—TUBERCULAR PHLEBITIS.

The section is from one of the pulmonary veins in a child dead of acute general miliary tuberculosis. Specimen loaned by Dr. W. P. Northrup.

Chronic periphlebitis produces thickening, principally of the outer coats of the veins, but the inner coats may also be involved. The surrounding tissue may be also thickened and coalesce with the walls of the vein. There may or may not be thrombosis.

Chronic endophlebitis is a not very common lesion, of the same general character as chronic endarteritis. More or less circumscribe patches of new connective tissue are formed in the inner coats, which may undergo fatty or calcareous degeneration.

Tubercular Inflammation of the walls of the veins may occur as an extension of the process from without or from a lodgment of the tubercle bacilli in the blood current on the intima (Fig. 151). This is not infrequent in the pulmonary veins, and Weigert has recently called attention to the fact that in acute miliary tuberculosis the growth of tubercle tissue into the lumina of these veins from tubercular lymph nodes is of frequent occurrence, and readily explains the topography and mode of occurrence of the general disease. The tubercle bacilli which are present in the tubercular tissue growing into the lumen of the veins find thus an easy distribution.

Syphilitic Inflammation may involve the walls of the veins either as gummy tumors or as more diffuse thickenings.

TUMORS.

Primary tumors of the veins are rare. Small *leiomyomata* have been described in the saphenous and ulnar veins. A *myo-sarcoma* as large as a man's fist has been described, situated in the dilated vena cava inferior. The veins are not infrequently secondarily involved by *sarcomata* and *carcinomata*, and sometimes by *chondromata*. The thin walls of the veins offer comparatively little resistance to the incroachment of malignant tumors, which thus gain access to the circulation and may form metastases in various parts of the body.

PARASITES.

Echinococcus is sometimes found in the veins, having either developed there or perforated from without.

Two species of *distoma* (*liver fluke*) occur in man. *D. hepaticum* occurs rarely in man, and while usually found in the bile ducts may occur in the vena cava. *D. hæmatobium* is very common in man in Egypt and in other parts of Africa, and usually occurs in the portal vein or its branches, and frequently in other veins.

THE CAPILLARIES.

The walls of the capillaries are so thin and so intimately connected with the surrounding tissues that their lesions are studied most appropriately among the diseases of the several organs. Dilatation of the new-formed capillaries in tumors, granulation tissue, etc., and fatty and hyaline degeneration of their walls, may be mentioned here as readily observed lesions occurring under a variety of conditions. The changes which we assume to occur in the walls of the smaller veins and capillaries in exudative inflammation, by reason of which fluids and blood-cells pass through them, are not yet sufficiently understood to be described with definiteness.

THE LYMPH VESSELS.

The smaller lymph vessels can hardly be treated as independent structures, since their walls are so closely joined with the tissues through which they pass; the lymph radicles, indeed, being nothing more than the spaces in the connective tissue in which the variously shaped connective-tissue cells lie. In the larger lymph vessels we find a moderate number of more or less independent lesions.

INFLAMMATION.

Lymphangitis.

Inflammation of the larger lymph vessels is usually secondary, and connected with some wound or injury. Owing, it is believed, to the entrance into the lymph trunk of some septic material or bacteria, the vessels, sometimes for a considerable distance away from the wound, become red, tender, and painful. Under these conditions, the microscopical appearances which the vessels present vary. In some cases, the redness disappears after death, and we find no appreciable alteration. In other cases, we find the walls of the lymph vessels more or less densely infiltrated with pus cells, and the lumen may contain variable quantities of pus and fibrin and desquamated endothelium. The tissue about the vessels may also be infiltrated with serum and pus. These lesions may undergo resolution and the vessel be restored to its normal condition; or the vessel wall and surrounding tissue may die or become involved in abscess; or new connective tissue may form in and about the vessel, sometimes with obliteration of its lumen. The lymph nodes may participate in the inflammatory process.

Inflammation of the lymph vessels may occur as the result of dissection and other wounds, and the bites of venomous reptiles. It may occur in the uterine lymphatics in the phlegmonous form of puerperal fever, and under other conditions.

Tubercular Lymphangitis.—Tubercular inflammation occurs both in large and small lymph vessels. Miliary tubercles and diffuse tubercle tissue may form in the walls and project into the lumen of the larger trunks; or in the smaller vessels the new growth may entirely fill the lumen, and grow in this, with more or less involvement of the walls. This may occur independently, but it is most frequently seen in connection with tubercular inflammation of adjacent tissues. Thus from tubercular lymph nodes in the vicinity of the thoracic duct there may be a direct extension of the tubercular inflammation, an involvement of the walls of the duct, and a growth of tubercle tissue into its lumen.

Such growths in the thoracic duct have been shown by Weigert to be frequent in acute general miliary tuberculosis, and very satisfactorily explain, on the hypothesis of the bacterial origin of the disease, the dissemination of the tubercles. In the vicinity of tubercular ulcers in the intestines, furthermore, we often see the subserous lymph vessels, which pass from the vicinity of the ulcers, distended with the products of tubercular inflammation, and looking like dense white knobbed cords.

Syphilitic Inflammation of the lymph vessels not infrequently occurs in the vicinity of syphilitic ulcers in the primary stage. In later stages there may be thickening of the walls of the vessels and the development of gummy tumors in and about them.

LYMPHANGIECTASIS.

Dilatation of the lymph vessels occurs under a variety of conditions. It may be congenital, or it may be due to some hindrance to the flow of lymph onward, as by pressure from any cause, or from the occlusion of the vessels by inflammation, or it may be produced by unknown causes. If the dilated vessels form a circumscribed mass, this is often called a *lymphangioma* (Fig. 67). In certain forms of *elephantiasis* and in *macroglossia,* the dilatation of the lymph vessels is an important factor. Its occurrence is not infrequent in the labia, prepuce, and scrotum.

TUMORS.

The relation of the endothelium of the lymph vessels and spaces to endotheliomata has been already mentioned in the section on Tumors.

The dissemination of malignant tumors through the lymph channels is of frequent occurrence, and is particularly marked in the case of carcinoma. In the vicinity of carcinomata, the lymph vessels are not infrequently crowded with the tumor cells, forming white, irregular cords; or small masses of the tumor cells may be found in the lymph vessels, either near to or remote from the tumor. White, irregular networks are often formed in this way beneath the pleura in carcinoma of the lung (Fig. 71), or beneath the capsule of the liver. Transverse sections of lymph vessels thus distended show sometimes swelling and detachment of the endothelium and a crowding of the lumen with tumor cells. Whether or not the endothelium participates in the new formation of the characteristic carcinomatous cells is not known.

THE LYMPH NODES (*Lymph Glands*).[1]

It is well, in studying the lesions of the lymph nodes, to remember that they are structures so placed in the course of the lymph vessels that the lymph, in flowing towards the larger central trunks, passes through them, undergoing a sort of filtration as it percolates through the trabeculæ of the lymph sinuses. If this simple fact be borne in mind, the diseases of the lymph nodes, which are, in the majority of cases, secondary, are much more readily understood. Particles of pigment which in any way get into the lymph vessels are carried along until a lymph node is reached, and here they are, in part at least, deposited among the trabeculæ of the sinuses, while the lymph passes on and out of the efferent vessels (Fig. 154). The same thing occurs when cells from malignant tumors, bacteria of various kinds, etc., gain access to the lymph vessels, and also, as there is good reason for believing, in the case of many poisonous materials which our present knowledge does not enable us to associate with bacteria. These various materials, filtered out of the lymph by the glands, may act in a variety of ways to produce lesions in them.

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[1] What we call *lymphatic tissue* embraces not only the so-called lymph glands and the less complex but still well-defined structures found in the stomach, intestines, tonsils, and elsewhere, and called lymph follicles, but also the less well-defined,

INFLAMMATION.

Acute Inflammation of the lymph nodes usually occurs in connection with some inflammatory process in the region from which its lymph is gathered. The nodes are in the majority of cases swollen, reddened, and softer than normal, and often the seat of smaller and larger hæmorrhages. Sometimes one, sometimes several nodes of a cluster are affected.

The microscopical examination shows the most prominent change to be a great increase in the number of cells in the follicles and cords, as well as in the lymph sinuses. These cells are, in part, small and spheroidal, and similar to those normally filling the meshes of the follicles; in part, large polyhedral or variously shaped cells, with prominent nuclei; the latter cells are most abundant in the lymph sinuses (Fig. 152). In addition to this, there is swelling of the endothelial cells of the reticulum of the sinuses. The blood-vessels may be distended with blood, or there may be blood, in greater or less quantity, free in the sinuses and follicles. The origin of the large number of new cells which may form in a very short time is not yet definitely known. They may

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irregular masses of tissue resembling that of lymph follicles, which, as *Arnold* has shown (Virchow's Archiv, Bd. 80, p. 315; Bd. 82, 394; Bd. 83, 289; Bd. 87, p. 114), is widely disseminated in variable amounts in different parts of the body; in the lungs, beneath the pleura and elsewhere; in the liver, kidneys, etc. Although the exact nature of these more diffuse masses of lymphatic tissue is too little understood, as indeed is that of the lymph follicles and glands themselves, there is reason to believe that they are analogous structures and prone to be affected by similar deleterious agencies. It seems better, in view of the fact that the so-called lymph glands are not glands at all, in the ordinary sense of the word, to call them *lymph nodes*, and the smaller masses of lymphatic tissue scattered through various parts of the body *lymph nodules* instead of "lymph follicles."

be emigrated leucocytes or their derivatives; they may be derivatives of the endothelium of the reticulum; or they may be in some cases, at least in part, cells which have been brought into the node, through the afferent trunks, from some external inflammatory focus. The capsule of the nodes, and not infrequently the connective tissue about them, may also be infiltrated with round cells.

Acute inflammation may terminate in resolution, the new cells disappearing either by fatty or other degeneration, or by being carried off in the lymph, and the node return to its normal condition. This is the rule in the less intense forms of inflammation. On the other hand, the inflammatory process may become purulent and so intense as to lead to the formation of abscess, usually with a greater or less involvement of the tissue about the nodes. There may be at first numerous small abscesses which coalesce to form larger ones. These abscesses—*buboes*—may open externally or internally, or they may become dried and converted into cheesy masses which may calcify and, by a chronic inflammation in their periphery, become inclosed by dense connective tissue. Sometimes, instead of abscess being formed, the tissue of the inflamed nodes becomes necrotic and breaks down, inducing more or less severe inflammatory or necrotic changes in the tissues in their vicinity.

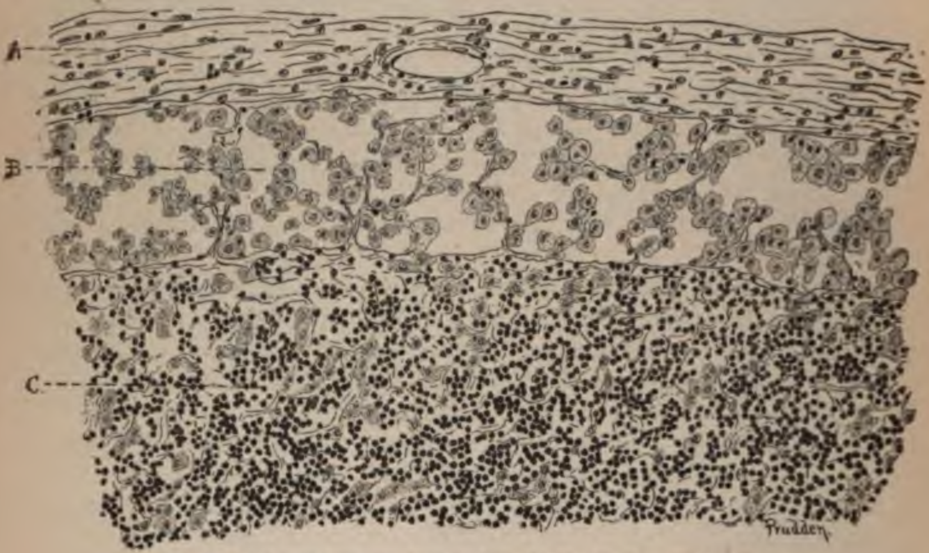

FIG. 152.—ACUTE INFLAMMATION OF LYMPH NODE IN TYPHOID FEVER.

Showing a portion of one of the mesenteric nodes. A, capsule; B, perifollicular space or lymph sinus, containing in its meshes many large cells; C, portion of one of the follicles, with large and small cells in the meshes of its reticulum.

In still other cases, acute inflammation of the lymph nodes passes into the chronic form.

Moderate degrees of inflammation in the lymph nodes are very common in connection with various forms of inflammation in neighboring parts. Thus simple pharyngitis, gastro-enteritis, erysipelas, simple purulent inflammation, etc., are often associated with this lesion of the nodes. The lymph nodes of children are, as a rule, more easily affected by moderate inflammations in neighboring parts than are those of adults. Purulent inflammation of the lymph nodes is most frequently associated with severer forms of inflammation of adjacent or related parts, especially those of an infectious character, syphilitic inflammation, poisoned wounds, pyæmia, etc. In a certain number of cases, we find bacteria in the inflamed lymph nodes, either singly or in zoöglœa colonies, which have presumably something to do with the lesion.

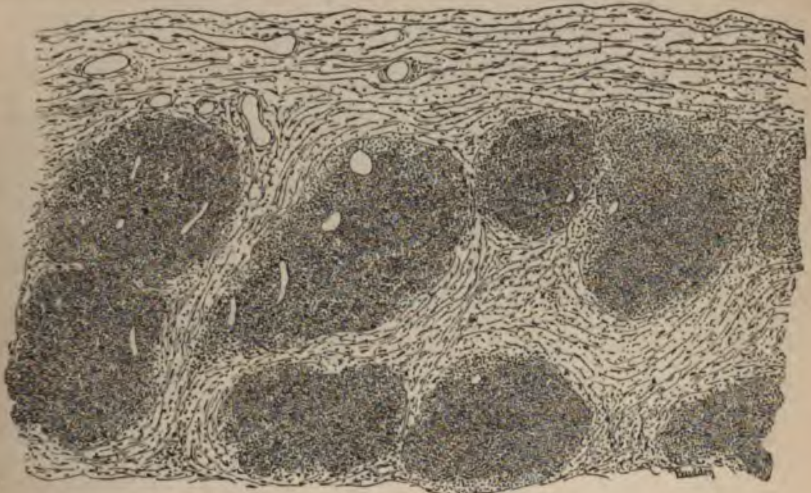

FIG. 153.—CHRONIC INFLAMMATION OF BRONCHIAL LYMPH NODE.

Showing obliteration of the lymph sinuses and atrophy of the lymph follicles by the new-formed connective tissue.

In many cases, the lesion of the lymph nodes appears to be induced, not by bacteria in the nodes themselves, but by ptomaïnes produced elsewhere by the action of bacteria and brought to the nodes by the lymph. The swelling of the lymph nodes in typhoid fever and diphtheria is probably induced in this way.

Chronic Inflammation.—This is characterized by an increase of the connective-tissue elements of the node, with a gradual and commensurate disappearance of the lymphoid cells. The reticulum of the follicles and sinuses becomes thickened and fibrous, and in the trabeculæ and capsule new connective tissue is formed until, in advanced cases, the entire node may be more or less completely converted into a mass of connective tissue. This condition is very frequently seen in the lower tracheal and in the bronchial nodes, apparently as a result of the lodgment in them of respired pigment particles; but it may occur in any nodes, either as a result of repeated moderate degrees of inflammation, or from causes which we do not know. In some cases, the nodes are greatly enlarged, and the new tissue contains many large cells, while in other cases the connective tissue is dense and contains but few cells (Fig. 153).

Pigmentation.—The pigment which is very frequently found in lymph nodes may be derived from the hæmoglobin of the blood, either in the nodes themselves or in remote parts, or it may be formed of various materials introduced into the body from without, such as the pigments used in tattooing, respired dust particles of various kinds—coal, stone, iron, etc. (Fig. 154). The pigment particles, which usually first lodge in the lymph sinuses, may collect here in large quantities, either in the reticulum or the cells lying in its meshes; they may penetrate the follicles and cords and find permanent lodgment there. They usually induce a greater or less degree of chronic inflammation, so that in extreme cases, such as are frequently seen in the bronchial lymph nodes, nothing is finally left of the node but a more or less deeply pigmented mass of dense connective tissue. The function of the node is, of course, in this way partially or entirely destroyed. The pigment in these cases appears to reach the node, in part by being carried along free in the lymph current, in part by becoming inclosed in leucocytes and being transported by them. Pigmentation of the nodes is most marked in those about the root of the lungs, which are frequently of a mottled

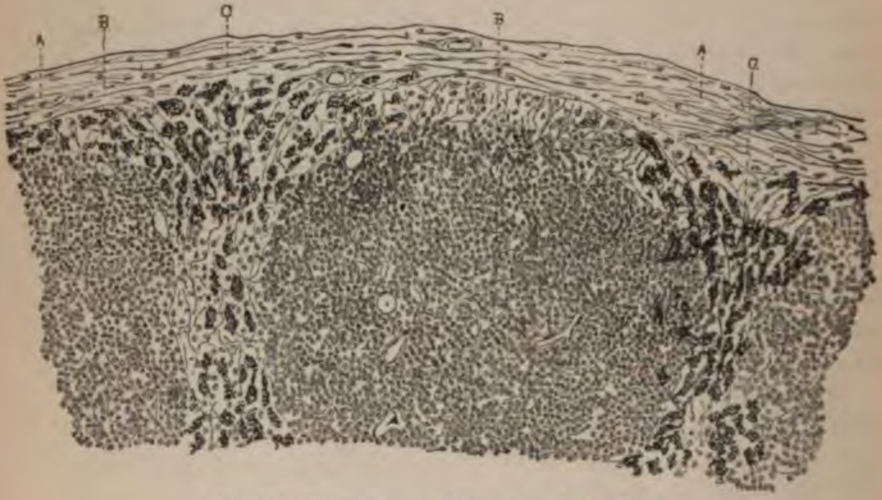

FIG. 154.—PIGMENTATION OF BRONCHIAL LYMPH NODE.

The pigment is largely in the lymph sinuses and inclosed in cells. A, capsule of node: B, lymph follicle; C, perifollicular lymph sinuses.

gray or a black color, but it may occur in the mesenteric and other nodes. Under similar conditions, the diffuse lymphatic structure in the lungs and liver may be similarly pigmented.

Inflammation of the Lymph Nodes with Cheesy Degeneration.—This lesion of the lymph nodes, which is distinct from the above-mentioned comparatively infrequent cheesy degeneration of the contents of old abscesses, commences with changes similar to those above described in simple inflammation. The node in this condition is swollen, and feels harder than normal; on section, it has a uniform reddish-gray color. Microscopical examination reveals a great increase in the number of parenchyma cells, some small and spheroidal, others large and polyhedral. Sometimes the larger cells are multinuclear, and not infrequently the reticular framework and the capsule are thickened. As the process advances, the characteristic necrotic changes make their appearance. We may find at first a greater or less number of the cells converted into a strongly refractile material, and the nuclei no longer capable of being stained. Then larger and smaller masses of cells undergo cheesy degeneration, with complete destruction of the blood-vessels, reticulum, and the spheroidal and other cells, and their conversion into a granular material. A section through the node in this condition shows the cut surface mottled with irregular-shaped, larger and smaller opaque white patches, which indicate the areas of cheesy degeneration. These patches may increase in size and coalesce, so that a large part of, or even the entire gland may be converted into a more or less dense cheesy mass which may be surrounded by the thickened capsule.

In this condition they may remain for a long time, and not infrequently, owing to the involvement of a series of associated nodes, either simultaneously or one after another, and the increase of connective tissue about them, we find large, irregular nodular masses made up of a congeries of similarly affected nodes.

On the other hand, the cheesy material may soften and break down, and, by the establishment of purulent and necrotic inflammation about them, abscesses may form which may open externally. These abscesses may heal; but usually the healing is difficult and slow, and long-continued suppurations, frequently with the development of fistulæ, are very common. Under these conditions, the inflammation may assume a tubercular character. Instead of softening, the cheesy material in the glands may become dry and hard, and undergo calcification.

Cheesy inflammation of the lymph nodes is most common in the cervical, bronchial, and mesenteric groups, but may occur anywhere. It is most apt to occur in badly nourished young persons, who, in addition to the lesion of the lymph nodes, are very liable to suffer from chronic inflammations of the mucous membranes, skin, periosteum, joints and the subcutaneous and other connective tissues. This general condition is known as *scrofula*, and the lesion of the nodes is sometimes called *scrofulous inflammation*. It is not infrequently associated with tubercular inflammation of the nodes, either as an independent lesion or as a part of a general tuberculosis, and by some writers tuberculous and scrofulous inflammation of the lymph nodes are considered to be identical. In a considerable proportion of cases, however, of so-called scrofulous inflammation of the lymph nodes, there is no formation of tubercle tissue, and we find no tubercle bacilli, so that we must consider this class of cases as simply inflammatory, with a tendency to cheesy degeneration.

Tubercular Inflammation may occur in connection with simple inflammatory changes in the lymph nodes, or with the form of inflammation which tends to cheesy degeneration. It may be local, confined to the nodes, or it may occur in connection with general acute miliary tuberculosis or with tubercular inflammation of single organs. It may occur in single nodes or in several nodes of the same group, or in groups situated in different parts of the body. In its simple and acute form, there may be no evident change to the naked eye in the appearance of the nodes, or they may be besprinkled with small grayish-white, translucent spots. Under these conditions, the nodes may be reddened and soft, or swollen and denser than normal. In more advanced forms of the lesion, the tubercles coalesce and undergo a greater or less degree of cheesy degeneration. Under these conditions, the cheesy areas are evident to the naked eye as more or less sharply circumscribed opaque whitish areas, frequently surrounded by an irregular, more translucent grayish zone of tubercle tissue, which merges insensibly into the adjacent tissue. The entire node may become involved, and more or less completely converted into a cheesy mass, in the periphery of which a zone of tubercle tissue may or may not be evident.

Microscopically the small nodules or miliary tubercles are seen to consist of more or less circumscribed collections of small spheroidal, or more frequently larger polyhedral cells, with or without well-defined giant cells. They usually commence to form in the follicles and lymph cords of the nodes, and from these may spread and involve the entire surrounding tissue. The cheesy degeneration, which here as elsewhere is apt first to involve the central portions of the tubercles, presents the usual appearances. Tubercle bacilli may be found in the edges of the cheesy areas, or in the tubercle tissue about them.

Simple inflammatory changes regularly occur in the periphery of the tubercles. There is an increase of cells in the lymph sinuses and follicles, and a more or less marked swelling, and apparently a proliferation of the cells of the reticular tissue of the node. In cases in which the process is chronic, there is often marked increase of the connective tissue of the nodes, the reticular tissue becomes dense and fibrous, and the trabeculæ and capsule are thickened. The tubercles themselves, instead of undergoing cheesy degeneration, may become fibrous or be converted into a hyaline material.

The cheesy material may dry and shrink, and become inclosed by a capsule of dense connective tissue and become calcified; or it may soften, and thus cavities be formed in the glands, filled with grumous material; or inflammatory changes may be induced in the vicinity of the nodes, leading to abscesses. On the other hand, hyperplastic inflammation in the periphery of the affected nodes may result in their becoming bound together into a dense nodular mass.

When cheesy degeneration has occurred, to the naked eye tuberculous lymph nodes may not be distinguishable from those in scrofulous inflammation, but in some cases the nodular character of the new tissue around the cheesy centres is evident. The process is usually a slow and chronic one, except when occurring in connection with acute miliary tuberculosis in other parts of the body. It may occur in any of the nodes, but is most frequent in those of the bronchial, mesenteric, and cervical regions.

Syphilitic Inflammation.—The lesions of the lymph nodes, which occur in connection with syphilitic poisoning, vary greatly, depending upon the stage of the disease. In the primary stage, the lymph nodes in the region of the seat of infection are apt to present the lesions of an ordinary acute inflammation, with a tendency to the assumption of the purulent form.

In the secondary stage of the disease, the nodes of other regions, neck, elbow, axilla, etc., are frequently swollen and hard. On microscopical examination, there may be an increase of connective tissue in the capsule and trabeculæ, but the chief change is in the accumulation in the follicles and lymph sinuses of larger and smaller spheroidal and polyhedral cells. The reticular tissue may be thickened, and the walls of the blood-vessels infiltrated with cells. In this condition the nodes may remain for a long time, not tending to form abscess; or they may undergo resolution through degeneration and absorption of the cells.

In the tertiary stage of the disease, the nodes may be the seat of chronic inflammation characterized by the formation of gummy tumors. Under these conditions, they may form large, firm nodular masses by the growing together by new connective tissue of several altered nodes. The gross and microscopical characters of gummata of the lymph nodes are, in the main, similar to those in other parts of the body.

There are important changes in the lymph nodes which occur as local manifestations of general diseases, such as *typhoid fever*, *leprosy*, etc., which will be considered under the headings of these diseases.

Degenerative changes in the lymph nodes, with the exception of th above described, are not of great frequency or significance.

Atrophy is a very regular occurrence in old age. In this condition, the nodes are small, hard, and, unless pigmented, white. Microscopical examination shows a marked diminution in the number of parenchyma cells, while the reticulum and the capsule and trabeculæ may be thickened. There may be an accumulation of fat around the node in connection with senile atrophy.

It should be remembered, in this connection, that the lymph nodes, as well as the lymphatic tissue in general, in children are more voluminous and contain a greater number of parenchyma cells than in adults.

Amyloid degeneration of the blood-vessels and reticulum of the lymph nodes occurs under the conditions which favor this change in general. It may occur in connection with amyloid degeneration of other parts of the body, or by itself. It may occur in nodes otherwise normal, or in those which are the seat of other lesions; thus in simple chronic or tubercular inflammation. It is frequently found in the mesenteric lymph nodes, in connection with waxy degeneration of the intestinal mucous membrane.

Hyaline degeneration of the external layers of the smaller arteries and the capillaries of the lymph nodes, and also of the parenchyma cells, occurs occasionally in old age or in connection with wasting diseases. The vessels and cells are swollen and converted into a translucent, strongly refractile substance resembling amyloid optically, but not responding to its micro-chemical tests. By the accumulation of this material, the uninvolved parenchyma of the nodes may be compressed and atrophied.

HYPERPLASIA OF THE LYMPH NODES (*Lymphoma*).

In addition to the considerable enlargements of the lymph nodes in inflammation which have been described above, they become enlarged under a variety of conditions which we do not understand. This lack of knowledge of the etiology, together with our ignorance of the function of the lymph nodes, and the morphological similarity, or even identity, which these enlarged nodes present, render it very difficult to decide upon the exact nature of the change, and in many cases to distinguish one form from another.

In the first place, there is a class of cases in which, sometimes slowly, sometimes with great rapidity, the lymph nodes of certain regions, especially the abdominal, axillary, cervical, and inguinal, enlarge not infrequently to an enormous extent. They may be either hard or soft, even almost fluctuating; the individual nodes may be distinct or merged into one another. Sometimes the nodes in nearly all parts of the body are affected. Microscopically we find that the enlargement is due, in the soft varieties, to an enormous increase of small spheroidal and polyhedral cells, and a growth of the reticular tissue. It is a new formation of lymphatic tissue, but the normal relations of follicles, cords, and lymph sinuses is not preserved. In the harder varieties, there is a thickening of the reticular tissue in addition to an increase of cells. In very rare cases, portions of the nodes may become cheesy. Sometimes larger and smaller hæmorrhages occur in the nodes, especially in the softer forms. In addition to these changes in the lymph nodes, there is, in a considerable proportion of cases, a new formation of lymphatic tissue in greater or less quantity in other parts of the body, in the spleen, in the gastro-intestinal canal, in the marrow of bones, in the liver, kidneys, etc., and the number of leucocytes in the blood and in other parts of the body is increased. This general condition is known as *leukæmia*, and will be considered under the general diseases. The enlarged lymph nodes in this disease may be called, for convenience, *leukæmic lymphomata.*

In the second place, there is a form of disease in many respects, particularly in the lesion of the lymph nodes, identical with leukæmia. There is, however, usually a less prominent involvement of the spleen and other lymphatic structures, and, what is more striking, no increase in the number of leucocytes in the blood. This is called *Hodgkin's disease*, or *pseudo-leukæmia*, and the enlarged lymph nodes may in this case be called *pseudo-leukæmic lymphomata.* The lesions of the lymph nodes are identical in both diseases, and it is convenient to assign different names to them simply because, for reasons which we do not at all understand, they seem to arise under different conditions, and to be associated with a constant difference in the character of the blood.

TUMORS.

Sarcomata occur in the lymph nodes as primary and secondary tumors, and these may be of various forms: spindle-celled, large and small round-celled, and angio-sarcomata. It is not easy in many cases to distinguish morphologically between the small round-celled sarcomata and the above-described lymphomata. *Fibromata, myxomata,* and *chondromata* occur in the lymph nodes, but are rare. Endotheliomata are described, but are not common. *Secondary carcinomata* are of frequent occurrence, the form of the cells and the nature of their growth depending upon the seat and character of the primary tumors.

PARASITES.

Aside from various forms of *bacteria* which are not infrequently found in the lymph nodes—thus in diphtheria, splenic fever, typhoid fever, tuberculosis, etc.—*filaria, trichinæ,* and *pentastomum* have been described.

THE ALIMENTARY CANAL.

THE MOUTH.

MALFORMATIONS.

Malformations of the lip and cheeks are usually associated with defective formation of the bones of the mouth. The entire process is generally due to an arrest of development.

1. The lower jaw is absent; the upper jaw and hard palate small and imperfectly formed; the temporal bones nearly touch in the median line. The lower part of the face is, therefore, wanting; the mouth is absent, or small and closed posteriorly; the tongue is absent. Such a malformation is rare; the fœtus is not viable.

2. The face remains in its early fœtal condition of a large cleft; the mouth and nose form one cavity; the orbits may be united in the same cavity. The fœtus is not viable.

3. There is a cleft in the upper lip, upper jaw, and hard palate. The cleft corresponds to the point of junction of the processes of the superior maxilla with the intermaxillary bone. There may be one cleft or two, one on either side of the intermaxillary bone. The cleft involves the lip alone, or the lip and superior maxilla, or the lip, maxilla, and palate. There may be a single or a double cleft in the palate; and the cleft may involve either the hard or soft palate, or both. If there are two clefts of the lip and maxilla, the portion of lip and bone between them may be small, or entirely absent, so as to leave a large open space. The soft palate may be entirely absent. This is a common malformation, and does not endanger life.

4. Rarely we find a cleft involving the middle of the lower lip, and sometimes extending into the inferior maxilla.

5. Either the inferior, the superior, or both maxillary bones may be abnormally small.

6. The edges of the lips may be partly or completely joined together. The opening of the mouth may be only a round hole.

7. The lips may be absent or imperfectly developed.

8. The corners of the mouth may be prolonged by clefts in the cheeks nearly to the ears.

HYPERTROPHY.

The skin of the cheeks and lips may be hypertrophied in connection with elephantiasis of the face.

There may be a thickening of the lips alone, so that they appear double. This thickening may be due to an increase of all the anatomical elements of the lips; or there may be an increase and dilatation of the lymphatic vessels, giving to the growth a soft, œdematous character.

INFLAMMATION.

Catarrhal Stomatitis is found most frequently in children. It is produced by a great variety of local and constitutional causes. Of the conditions which are seen during life, the congestion, increased production of mucus, and swelling of the mucous membrane, but little remains after death.

During life, the congestion and swelling of the mucous membrane are well marked. There are often white patches, produced by the death of the superficial epithelial cells. There may be an increased production of mucus, which runs constantly from the mouth, or, instead of this, the entire mucous membrane is unnaturally dry.

The only structural changes which can be demonstrated are the degenerative changes of the epithelial cells and the production of pus globules, which infiltrate to a moderate degree the stroma of the mucous membrane and appear on its surface.

Croupous Stomatitis is produced by local irritants, by extension of the same form of inflammation from the pharynx, and it occurs with the exanthematous fevers and with diphtheria.

Portions of the mucous membrane are swollen and congested, and covered with a false membrane. This false membrane is composed of a thickened layer of epithelium in the condition of coagulation necrosis, and of fibrin and pus in variable relative quantity. The stroma of the mucous membrane is infiltrated with pus and fibrin, and portions of it may become necrotic.

STOMATITIS ULCEROSA[1] (*Stomacace; Stomatite Ulcero-membraneuse*).

This form of stomatitis occurs in children between the ages of four and eight years, and in adults between the ages of eighteen and twenty-five years. It is apt to occur in localized epidemics, in hospitals and asylums, and among soldiers and sailors. Some of the forms of mercurial stomatitis seem to be identical with this form of inflammation.

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[1] *Bergeron*, "Stomatite ulcerosa." Union Médicale, 1859. *Bohn*, "Mundkrankheiten der Kinder," 1880.

The inflammation begins at the margin of the gums of the lower jaw. The gums are swollen and coated with a grayish, soft matter, composed of bacteria and detritus. Then follows destruction of tissue; the gums are destroyed around the teeth, and these fall out; the inflammation extends to the lips, cheeks, and tongue. The ulcers are coated with a thick, soft, gray membrane. The surrounding soft parts are swollen, and there may be necrosis of the jaws.[1]

Syphilitic Stomatitis.—As a result of syphilis, there may be produced either the so-called mucous patches or gummy tumors. In the mucous patches, we find at first the epithelial layer thickened and the papillæ of the stroma swollen and infiltrated with cells. This may be followed by desquamation of the epithelium and ulceration of the stroma.

The deeper gummy tumors may also soften and form ragged ulcers of some size.

Tubercular Stomatitis commences with the formation of miliary tubercles or of larger tubercular masses in the stroma of the mucous membrane. These masses soon degenerate, soften, and form ragged ulcers resembling very closely syphilitic ulcers.

GANGRENE.

Gangrene of the lips and cheeks, or noma, is most frequent in cachectic children as a consequence of the abuse of mercury. Much more rarely it occurs in adults after typhus and other exhausting diseases. The disease begins in the mucous membrane of the cheeks near one of the corners of the mouth. The mucous membrane becomes black and gangrenous; the gangrene extends rapidly through the entire thickness of the cheek, and produces perforation; it extends laterally in all directions.

TUMORS.

Adenomata are formed in the mucous membrane covering the mouth, lips, and soft palate. The tumors are rounded, usually small, sometimes as large as a hen's egg. They may be situated in the thickness of the mucous membrane, or project in a polypoid form. They are formed by an hypertrophy of the normal mucous glands. The glandular acini are increased in number and size, the epithelial cells are increased in number and may undergo colloid degeneration.

Papillomata occur most frequently at the edges of the lips, but are also found on the gums, the floor of the mouth, and the cheeks. They are formed of hypertrophied papillæ, covered with thickened epidermis. They very often ulcerate.

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[1] *R. Volkmann,* Virch. Arch., Bd. 50, p. 142, describes five cases of inflammation of the mucous glands of the lower lip. The lip was swollen and hard, the mucous glands and their ducts were dilated.

Carcinomata are of frequent occurrence. They may be found at any part of the mucous membrane of the mouth, but as a rule begin in the edge of the lower lip.

They may originate in an ulcerating papilloma, or as a flat, superficial growth from the deeper layers of the epithelium, or as deep nodules starting in the mucous glands. They are composed of large masses of epithelial cells, closely packed together, often forming nests, and arranged in anastomosing tubular masses. The stroma surrounding these masses is infiltrated with cells. In a few cases the infiltration of the stroma with small round cells may be very marked, so marked that the epithelial growth may be obscured. The new growth increases in size, ulcerates, infiltrates the adjacent tissues, and may give rise to metastatic tumors.

Angiomata are found in the lips. They may be congenital or developed after birth.

Fibromata, lipomata, and *enchondromata* have been seen in a few cases in the lips. When they appear in the mouth they usually grow from the bones.

THE TONGUE.

MALFORMATIONS.

Absence of the tongue is found in connection with the extreme defects of development of the face already mentioned.

The anterior portion of the tongue may be absent while its base remains. The lower jaw is then small.

The tongue may be partly or completely adherent to the floor of the mouth. The frenulum may be abnormally short, or may extend to the tip of the tongue. In rare cases, the sides of the tongue are adherent, or its upper surface may be adherent to the roof of the mouth.

HYPERTROPHY.

Macroglossia, or hypertrophy of the tongue, is almost always a congenital lesion, and is especially common in cretins. The tongue is so large that the cavity of the mouth cannot contain it; it is protruded through the lips and displaces the jaws. The lips may also be hypertrophied in the same way.

There is an hypertrophy of all the anatomical elements which make up the tongue, and in addition to this there may be a dilatation of the lymphatic vessels.

INFLAMMATION.

Inflammations of the tongue may be associated with similar changes in the mouth, or may occur by themselves.

Superficial Glossitis.—Inflammation involving only the mucous membrane of the tongue may occur as an acute or chronic process.

The acute forms present no marked lesions.

The chronic forms result in an increased production of epithelium and an hypertrophy of the papillæ of the tongue.

A moderate development of such an inflammation is not infrequently associated with derangements of the stomach. The tongue is large, its surface is irregular from the hypertrophy of the papillæ. There may be no change in the epithelium, and then the surface of the tongue is clean and red; or the epithelium is increased and the tongue is covered with a white fur.

More severe forms of the disease also occur, especially with syphilis. The hypertrophied papillæ and increased epithelium then alter very decidedly the appearance of the tongue.

Parenchymatous Glossitis may be produced by mercurial poisoning, by injury, or by unknown causes. The tongue is swollen, the muscular and connective portions are congested and infiltrated with serum and pus. The inflammation may stop at this point, or it may go on to the formation of an abscess.

Syphilitic Glossitis.—In persons suffering from constitutional syphilis there may be mucous patches on the surface of the tongue; or gummy tumors in its stroma, which often soften and form deep ulcers; or a diffuse, chronic inflammation of the surface of the tongue, with hypertrophy of the papillæ.

Tubercular Glossitis.—There may be a tubercular inflammation of the connective tissue of the tongue just beneath the epithelial layer, resulting in the formation of tubercle granula and granulation tissue. In this way tumors of some little size are formed, which may remain unchanged for some time, or may degenerate, soften, and form ulcers.

TUMORS.

Cysts.—The most common forms of cysts are the sacs beneath or partly in the substance of the tongue (ranula). They are formed by dilatation of the ducts of the submaxillary and sublingual glands; or make their appearance in the connective tissue beneath and in the tongue.

Angioma.—Cavernous vascular tumors are found in the substance of the tongue and projecting from its surface.

Lipoma and *fibroma* are rare. They form nodules in the substance of the tongue, or project in a polypoid form. Composite tumors, composed largely of fat, are found in the tongue as a congenital condition.

Lupus occurs in the form of nodules and ulcers at the base of the tongue.

Sarcomata are rare in this situation. I (Delafield) have seen one such tumor in a young child. It formed a nodule as large as a chestnut, and was composed of round and fusiform cells.

Carcinoma.—This form of new growth may begin in the tongue or may extend to it from the adjacent tissues. The growth is composed of large, flat epithelial cells packed closely together in anastomosing tubular spaces and surrounded by a connective-tissue stroma.

THE PHARYNX AND THE ŒSOPHAGUS.

MALFORMATIONS.

Fistulous openings from the pharynx, running between the muscles and perforating the skin, have been seen in rare instances. These fistulæ are so small as to have no practical interest.

The œsophagus may be entirely absent, or its lower portion may be present and joined to the pharynx by a solid cord; or the pharynx, or the lower part of the œsophagus, may be continuous with the trachea; or the entire œsophagus may be represented by a solid cord.

Diverticula of the pharynx, dilatations of the œsophagus, and division of the middle portion of the œsophagus into two branches have all been observed.

INFLAMMATION.

Catarrhal and Croupous Pharyngitis are usually associated with the same forms of inflammation in the mouth, and have the same characters.

Submucous Pharyngitis may occur with inflammations of the mucous membrane, with caries of the cervical vertebræ, with inflammation of the cervical and parotid glands, with periostitis of the cranial bones, or may be idiopathic. It may result in swelling and œdema, in induration, or in suppuration. It is most important when it affects the posterior wall of the pharynx and forms retro-pharyngeal abscesses. Such abscesses may cause death by suffocation.

Catarrhal Œsophagitis may be either acute or chronic. The chronic form may produce ulceration, or relaxation and dilatation of the walls, or hypertrophy of the muscular coat.

Croupous Œsophagitis is found with croup of the pharynx, and after the exanthemata and other severe diseases.

Irritating and caustic acids and alkalies destroy larger or smaller portions of the mucous membrane. The necrosed portions are of a black or whitish color, surrounded by a zone of intense congestion. If the patient recover, the patches of membrane which have been destroyed slough, fall off, and leave a granulating surface. In this way, dangerous stenosis of the œsophagus may be produced.

Foreign bodies which are swallowed and become fixed in the œsophagus cause inflammation of the mucous membrane and of the adjoining soft parts. The inflammation may go on to produce abscesses around the œsophagus, or to destroy the wall of the canal, and the foreign body finds its way into the trachea, aorta, or pericardium.

Inflammation of the submucous tissue of the œsophagus, apart from the cases just mentioned, is not common. It may cause the formation of abscesses, or of fibrous tissue, which may produce stenosis.

ULCERATION.

Ulceration of the pharynx occurs in rare cases as the result of catarrhal inflammation. More frequently it is produced by syphilis, either in the form of superficial ulcers or of deep and extensive destructions of tissue from the softening of gummy tumors.

Lupus also sometimes attacks the upper part of the pharynx, and produces extensive ulceration. Ulceration of the œsophagus is not common, but a few cases of simple perforating ulcer have been described.[1]

Foreign bodies in the œsophagus may perforate its wall, as already mentioned. Perforation of the œsophagus from without may be produced by inflamed bronchial glands, by cavities and gangrene of the lungs, by abscesses in the mediastinum, by abscesses accompanying caries of the vertebræ, and by aneurisms of the aorta. Cases have been described of rupture of the wall of the œsophagus by violent coughing and vomiting, but it seems probable that there was really some previous disease to account for the rupture.

DILATATION.[2]

Simple cylindrical dilatation of the œsophagus is usually the result of long-continued stenosis of the œsophagus or of the cardiac end of the stomach; although not nearly all the stenoses are followed by dilatation. These dilatations are formed at first immediately above the stenosis and then extend upward. Only in rare cases does the dilatation involve the whole length of the tube. The entire wall of the dilated portion of the œsophagus is thickened, and there may be polypoid outgrowths from the mucous membrane.

In rare cases there is cylindrical dilatation of part or of the whole of the œsophagus without a stenosis or any discoverable cause. In these cases the dilatation is usually greatest near the middle of the œsophagus and diminishes upward and downward, so that the œsophagus has a fusiform shape. The dilatation may reach a very considerable degree, the walls of the œsophagus are thickened, its mucous membrane may be covered with papillary outgrowths or ulcerated.

The Sacculated Dilatations of the œsophagus are of two kinds: those due to pressure, and those due to traction.

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[1] *Graefe u. Walther*, Jour. f. Chir. u. Augenheilk., Bd. 19. Med. Chir. Trans., Vol. 36. *Rokitansky*, "Path. Anat."

[2] *Ziemssen*, "Cyclopædia of Medicine," viii., p. 47.

The dilatations due to pressure are situated in the posterior wall of the pharynx, just at its junction with the œsophagus. The smaller sacs are from the size of a pea to that of a hazelnut; the larger sacs may reach an enormous size, and hang down between the œsophagus and the vertebral column, the opening into the œsophagus remaining comparatively small. It is supposed that a limited area of the wall of the œsophagus loses its power of resistance against the pressure exercised upon it in each act of swallowing; it then is forced outward by the pressure, and so there is formed first a protrusion and then a sac. When a sac is formed, the food enters it, accumulates there, and so the sac becomes larger and larger.

The dilatations due to traction are situated on the anterior wall of the œsophagus, at a point nearly corresponding to the bifurcation of the trachea. They are of funnel shape, with the small end outward. Their length varies from two to twelve millimetres; the width of the opening into the œsophagus is from six to eight millimetres.

These dilatations are due to inflammation of the parts adjoining the œsophagus, especially of the bronchial glands, followed by adhesions to some part of the anterior wall of the œsophagus. These adhesions then contract and draw the wall of the œsophagus outward, and in this way the dilatations are formed.

At a later time these sacs may perforate into the bronchi, the lungs, the pleural cavity, the pericardium, the aorta or pulmonary artery.

STENOSIS.

Congenital Stenosis.—Besides the defects of development of the œsophagus which are incompatible with life, there may be a congenital stenosis of some part of it which causes difficulty in swallowing, but yet does not destroy life.

Stenosis by Compression is not uncommon. Tumors of the neck and mediastinum, and aneurisms of the aorta, are the usual causes.

Stenosis by Obstruction.—Foreign bodies may be lodged in the œsophagus. Tumors may hang down from the pharynx into the œsophagus, or may be situated in the wall of the œsophagus. Inflammation of the œsophagus, due to the ingestion of irritating poisons, produces cicatricial stenoses. A few cases of stenosis due to syphilitic inflammation have been reported.

TUMORS.

The veins of the œsophagus may be enormously dilated. They may rupture, and so give rise to hæmorrhage.[1]

Cysts.—Small retention cysts of the follicles of the mucous membrane are sometimes found. Van Wyss[1] describes a cyst as large as an apple attached to the posterior wall of the œsophagus, one and one-half inches above the stomach. It was filled with ciliated epithelium.

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[1] *Bristowe*, Trans. London Path. Soc., 1856.

Papillomata of small size may be found in considerable numbers throughout the entire length of the œsophagus, or may occur singly. Large papillary tumors are more rare.

Fibromata grow from the periosteum of the bones at the base of the skull, and project into the cavity of the pharynx and posterior nares in the form of large polypoid tumors. Small fibrous tumors are formed in the submucous connective tissue of the œsophagus. Tumors, which attain a very large size, originate in the submucous connective tissue on the anterior wall of the lower part of the pharynx, and as they grow hang down into the œsophagus.

Lipomata of small size are sometimes found in the wall of the œsophagus.

Myomata composed of smooth muscle may grow in the muscular coat of the œsophagus and attain a considerable size.[2]

Carcinomata may originate at any part of the wall of the pharynx and œsophagus. They are composed of flat epithelial cells closely packed together in masses in the usual way. In the œsophagus, the new growth begins in the deeper layers of the mucous membrane, and grows so as to encircle the tube for a length of one or more inches. The tumor remains as a flat infiltration, or it ulcerates, or it projects inward in large fungous masses. The growth may extend up and down the œsophagus and even involve the pharynx or stomach.

The ulcerative process may extend outward so as to produce perforation into the air passages, the lungs, pleuræ, pericardium, and large blood-vessels.

The new growth may extend outward and infiltrate the surrounding soft parts, so that the œsophagus is surrounded by large, solid, cancerous masses. Metastatic tumors are also sometimes formed.

Adenoma.—A polypoid adenoma composed of tubules lined with cylindrical epithelium, and growing from the anterior wall of the œsophagus, has been described by Weigert.[3]

I (Delafield) have seen one tumor, the size of a chestnut, growing in the soft palate, which was composed of a stroma of connective and mucous tissue in which were irregular, anastomosing tubules filled with small, polygonal, nucleated cells. It could be called an adenoma or a carcinoma.

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[1] Virch. Arch., Bd. 51, p. 143.

[2] Virch. Arch., Bd. 43, p. 137. Med. Times and Gazette, Nov. 28th, 1874. Glasgow Med. Journal, Feb., 1873.

[3] Virch. Arch., Bd. 67, p. 516.

Another composite tumor grew from the mucous membrane of the pharynx behind the left tonsil. It filled the pharynx below the level of the palate. It had the gross appearance of a myxo-sarcoma, the central portions being very soft. It was composed of connective tissue, mucous tissue, fat, sarcomatous tissue, and irregular tubules lined with small, polygonal epithelial cells. Some of the tubules were distended with masses of hyaline matter. The whole structure resembled that of the tumors so often found in the parotid region—tumors which can be called "adenoid myxo-sarcomata."

Sarcoma.—I have seen one case in which there was a diffuse growth involving both the tonsils, the posterior and lateral walls of the pharynx, the base of the tongue, and the epiglottis. The new growth replaced the mucous membrane, infiltrated the soft parts for a short distance, and projected inward in polypoid masses. It was composed of small, polygonal, nucleated cells, contained in a very delicate nucleated stroma (Fig. 155).

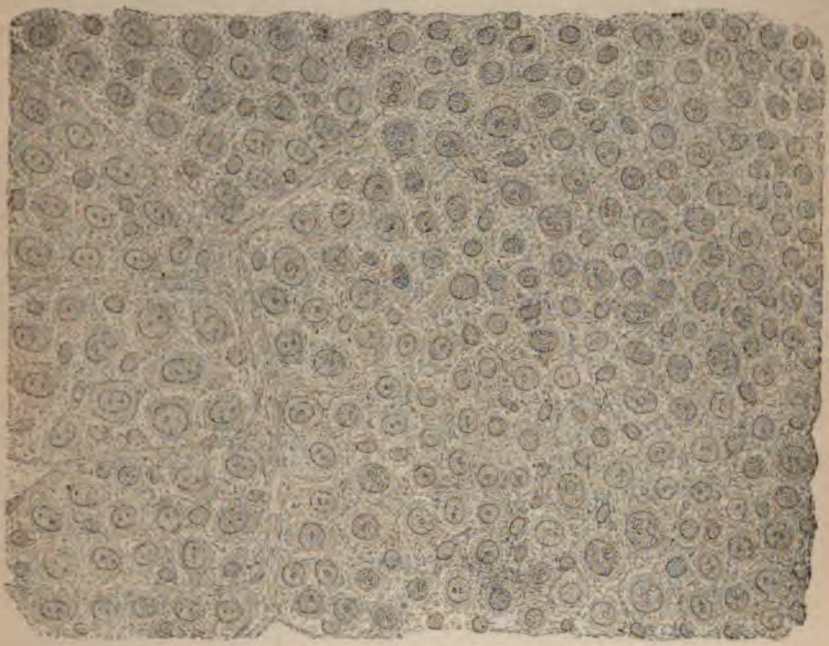

FIG. 155.—DIFFUSE SARCOMA OF THE PHARYNX, × 850 and reduced.

THE STOMACH.

MALFORMATIONS.

Malformations of the stomach are not common. The organ may be entirely wanting in acephalous fœtuses. It may be of various degrees of smallness, sometimes no larger than the duodenum. It may be divided into two halves by a deep constriction in the middle. The pyloric orifice may be stenosed or entirely closed. The stomach may be outside of the abdominal cavity from a hernial protrusion through the diaphragm, or at some point in the abdominal wall. It is found on the right side, instead of the left, when the other viscera are transposed, and the position of the cardiac and pyloric orifices is correspondingly inverted.

POST-MORTEM CHANGES.

In adults, the stomach after death is of a grayish or pinkish color, sometimes mottled with red ecchymoses. The mucous membrane is soft, and the epithelium easily brushed off. At the fundus the food is usually found collected, and here the mucous membrane is the softest. It is very common to find the epithelium removed from the entire fundus of the stomach, so that all that portion of its wall is grayer and thinner, there being a sharp dividing line between the two portions. Sometimes this post-mortem softening process goes on to destroy all the coats of the stomach, and even the adjoining portion of the diaphragm. In this way, the contents of the stomach may be emptied into the pleural cavity by a large, ragged opening in the stomach and diaphragm. When the softening affects all the coats of the stomach, the softened portion is not sharply limited. The entire thickness of the affected portion of the wall is converted into a gray or yellow semi-transparent jelly, or into a blackish, broken-down pulp.

This softening is most frequent in children, but also occurs in adults, usually in connection with severe and exhausting diseases.

A similar post-mortem softening of the wall of the œsophagus has been described by Moxon.[1]

INJURIES.

Perforating wounds of the stomach usually give rise to a fatal peritonitis. It is possible, however, for the wound to heal, or a gastric fistula may be formed.

Rupture of the stomach may be produced by severe blows or falls.

HÆMORRHAGE.

Small extravasations of blood in the wall of the stomach are frequently found in persons who have died from one of the infectious diseases.

Hæmorrhage into the cavity of the stomach may be produced in a variety of ways.

In ulcers of the stomach, there may be bleeding from the small vessels of the ulcer, or from the perforation of a larger artery.

In cancer of the stomach, there may be bleeding from the tumor.

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[1] Trans. Lond. Path. Soc., 1870, p. 159.

Some cases of chronic gastritis are characterized by general bleeding from the mucous membranes of the stomach.

Cirrhosis of the liver is not infrequently attended with large hæmorrhages from the mucous membrane of the stomach.

Small aneurisms of the arteries in the wall of the stomach may rupture internally.

In yellow fever and some of the other infectious diseases, there is hæmorrhage into the cavity of the stomach.

Patients may vomit blood during life, and after death no lesion to account for the bleeding be found.

INFLAMMATION.

Acute Catarrhal Gastritis, as we see it after death, is usually due to the ingestion of irritating substances, or forms part of the lesions of cholera morbus. If we can judge from clinical symptoms, it occurs during life as a temporary condition from a variety of causes.

After death the mucous membrane is found congested and swollen, or the congestion may have disappeared. The mucous membrane is coated with an increased amount of mucus, especially at the pyloric end of the stomach. Sometimes there are a number of minute white dots in the substance of the mucous membrane.

The structural changes in the mucous membrane consist simply in a swelling of the cells of the gastric tubules, a slight infiltration of the stroma with pus cells, and a swelling of the patches of lymphatic cells. The little white dots, when they are present, are composed of small foci of pus between the gastric tubules, with degeneration and destruction of some of the tubules.

Chronic Catarrhal Gastritis is a very common disease. There is, however, no very close relation between the severity of the symptoms during life and the extent of the lesions found after death.

In some cases, chronic alcoholism, or the abuse of drugs, or the mode of life of the patient seems to be the cause of the lesion. Chronic phthisis, chronic Bright's disease, cirrhosis of the liver, and fatty liver are often accompanied by chronic gastritis. Organic disease of the heart, or pressure on the ascending vena cava, produces a form of chronic gastritis characterized by intense general congestion.

After death the stomach is found either empty or still containing food. It is of normal size, or dilated, or small, sometimes hardly larger than the duodenum. Its inner surface is coated with a thick layer of tenacious mucus, most abundant at its pyloric end. The mucous membrane is congested, or white, or slate colored, or mottled with small white spots. It is of normal thickness, or thinned, or thickened, or there are little polypoid projections from its surface, or there is cystic dilatation of the gastric tubules. The connective tissue and muscular coats remain unchanged, or they are thinned and relaxed, or they are hypertrophied. The hypertrophy may be diffuse, or it is confined to the pyloric end of the stomach and may then produce stenosis of the pylorus.

The minute lesions consist principally in changes in the mucous membrane. The cells of the gastric tubules are swollen, degenerated, and broken down. The tubules are atrophied and deformed, or dilated into cysts. The patches of lymphatic tissue about the blind ends of the tubules are increased in size. The connective tissue between the tubules is infiltrated with cells and increased in quantity.

Croupous Gastritis is of rare occurrence. It is found in children with croupous inflammation of the pharynx and œsophagus, and is then usually in small patches. In adults, it is almost always secondary to typhus, pyæmia, puerperal fever, cholera, dysentery, the exanthemata, and irritating poisons. The false membrane is in small patches, or may line a large part of the stomach. The disease is usually not diagnosticated during life, the symptoms of the primary disease diverting attention from the gastritis.

I (Delafield) have seen one case of idiopathic croupous gastritis in an adult. A man, forty-six years old, was in good health until eight days before his death. At that time he caught cold, had pains over his bowels, tenderness over the liver, constipation, cough with mucous expectoration, temperature 102½°, pulse 120. On the day of his death, the eighth day of the disease, the temperature was 100°, pulse 112, tongue dry, abdomen tympanitic and tender, and he died in a prolonged attack of syncope. At the autopsy all the viscera were examined. Excepting evidences of bronchitis in the lungs, there were no lesions save in the stomach. About two-thirds of the internal surface of the stomach, including the lesser curvature and anterior and posterior walls, appeared to be covered with a thick false membrane, which did not quite reach to the cardiac or pyloric orifices. Minute examination showed that there was a layer of exudation on the internal surface of the mucous membrane. This exudation consisted of fibrillated fibrin and lymphoid cells dipping into the mouths of the follicles. Beneath the exudation the mucous membrane was thickened and altered. A large number of lymphoid cells separated the follicles, and even replaced them entirely. The submucous layer was very much thickened by the presence of lymphoid cells, fibrillated fibrin, and fibrous tissue. The muscular coat was separated into layers by groups of lymphoid cells.

Wilks and Moxon[1] mention a similar case in a man with chronic Bright's disease; and a case of both croupous gastritis and colitis with abscess of the liver.

Suppurative or Phlegmonous Gastritis.—A formation of circumscribed collections of pus may occur in the connective-tissue coat of the stomach, as it does in other parts of the body, in puerperal fever and the infectious diseases.

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[1] "Path. Anat.," p. 381.

Idiopathic suppurative gastritis is a disease of rare occurrence. Leube[1] has collected 31 cases, of which 26 were males and 5 females. In some of the cases the inflammation was ascribed to the excessive use of alcohol, in others to a wound in the region of the stomach, in others to some error in diet.

Fagge[2] describes a case in a male of fifty-one years of age, without discoverable cause.

Silcock[3] describes a case in which the gastritis followed the operation of gastrostomy.

I have seen one case occurring in an adult male, without any known cause.

The suppurative inflammation seems to begin in the connective-tissue coat of the stomach. From thence it may extend to the glandular coat and produce perforations, or outward to the muscular and peritoneal coats. In some cases there is added a local or general peritonitis.

The inflammation may involve one or more circumscribed areas and so produce abscesses, or it may be a diffuse process involving the whole extent of the wall of the stomach.

Toxic Gastritis.—The mineral acids, the caustic alkalies, arsenic, corrosive sublimate, and the metallic salts, phosphorus, camphor, and all other irritating materials, cause different lesions of the stomach, according to their quantity, their strength, and the length of time that has elapsed before death.

In large quantities, they destroy and convert into a soft, blackened mass both the mucous membrane and the other coats, so that perforation may take place. In smaller quantities, they produce black or white sloughs of the mucous membrane, surrounded by a zone of intense congestion. If death does not soon ensue, the ulcerative and cicatricial processes which follow such sloughs may contract and deform the stomach in various ways.

If the poisons are of less strength, they produce a diffused congestion of the mucous membrane, with catarrhal or croupous exudation on its surface, and serous infiltration of the submucous coat (see chapter on Poisons).

ULCERS OF THE STOMACH.

The Chronic Perforating Ulcer.—This form of ulcer is often seen; according to Brinton, in five per cent of persons dying from all causes.

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[1] *Leube*, "Ziemssen's Cyclopædia," vii., p. 157.

[2] Trans. Lond. Path. Soc., 1875, p. 81.

[3] Trans. Lond. Path. Soc., 1883, p. 90.

It occurs in females nearly twice as frequently as in males. As regards the age, Brinton concludes that the liability of an individual to become the subject of gastric ulcer gradually rises, from what is nearly a zero at the age of ten, to a high rate, which it maintains through the period of middle life; at the end of which period it again ascends, to reach its maximum at the extreme age of ninety. Lebert gives one hundred and ninety-eight cases in which the ulcers were found at the autopsy, as follows:

AGE.	NUMBER OF CASES.	AGE.	NUMBER OF CASES.
15 to 20 years	20	50 to 60 years..........	29
20 to 30 years..........	48	60 to 70 years..........	19
30 to 40 years.........	28	70 to 80 years..........	5
40 to 50 years..........	43		

Hauser[1] gives thirty autopsies from Erlangen of ulcers which were still open, as follows:

AGE.	NUMBER OF CASES.	AGE.	NUMBER OF CASES.
20 to 30 years..........	3	50 to 60 years..........	7
30 to 40 years..........	3	60 to 70 years..........	8
40 to 50 years..........	3	70 to 80 years..........	6

Moore[2] gives the following table of the fatal cases of ulcer of the stomach occurring at St. Bartholomew's Hospital from 1867–1879:

SEX.	AGE.	POSITION.	CAUSE OF DEATH.
M.	36	Near pylorus..	Perforation.
M.	19	Greater curve near pylorus	Hæmorrhage.
M.	47	Near pylorus..	Exhaustion.
M.	47	Pylorus...........	Phthisis.
M.	41	"	"
M.	52	"	Exhaustion.
M.	46	Lesser curve near pylorus	Perforation.
F.	47	....	Sinus in liver to lung.
M.	57	Cardiac end..................	Hæmorrhage.
M.	19	Near pylorus	Perforation.
M.	40	"	"
F.	46	Posterior wall..................	"

Goodhardt[3] describes an ulcer of the stomach, which proved fatal, from hæmorrhage in an infant at birth.

The situation of these ulcers, according to Brinton, is as follows: In 43 per cent, the posterior surface; in 27, the lesser curvature; in 16, the pyloric extremity; in 6, both the anterior and posterior surface; in 5, the anterior surface only; in 2, the greater curvature; in 2, the cardiac pouch. Thus about 86 ulcers in every 100 occupy the posterior surface, the lesser curvature, and the pyloric sac.

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[1] Das chron. Magengesch., 1883.

[2] Trans. Lond. Path. Soc., 1880, p. 110.

[3] Trans. Lond. Path. Soc., 1881, p. 79.

As regards the number of ulcers, two or more are present in about twenty-one per cent; there may be two, three, four, or even five ulcers. In cases of multiple ulcers, the ulcers are often developed successively.

In size, the ulcers vary from one-quarter of an inch to five or six inches.

They are usually of circular shape, sometimes oval; sometimes two or more are fused together.

The perforation is largest in the mucous membrane. It may remain confined to this, or extend outward and involve the connective tissue, muscular and peritoneal coats, its diameter becoming smaller as it advances. The ulcer looks like a clean hole punched out of the wall of the stomach. Its floor shows no active inflammatory changes. Its edges may be in the same condition, or they may be thickened by the growth of connective tissue and cells. The rest of the mucous membrane of the stomach is apt to be in a condition of chronic catarrhal inflammation.

The ulcer may perforate directly through the wall of the stomach, and the contents of the latter are discharged into the peritoneal cavity; or adhesions are formed between the wall of the stomach and the neighboring viscera, so that the bottom of the ulcer is closed; or, if the liver, the intestines, or the abdominal wall become adherent, they may be invaded by the ulcerative process, and cavities or fistulæ are formed communicating with the stomach; or, if the adhesions are incomplete, a local peritonitis and collections of pus may be developed.

During the progress of the ulcer, there may be repeated small hæmorrhages from the erosion of small blood-vessels, or large hæmorrhages from the erosion of large arteries.

In many cases these ulcers cicatrize, and such a cicatrization may produce various deformities of the stomach.

It is very difficult to understand how these ulcers are produced. It seems probable that the nutrition of a circumscribed part of the wall of the stomach is interfered with, and that this portion is then destroyed by the action of the gastric juice. But we are still ignorant of the way in which the obliteration of the arteries is effected. It has, indeed, been demonstrated in animals that an artificial embolism of the branches of the gastric arteries will produce ulcers of the stomach; and in the human stomach we occasionally meet with cases of embolism of the branches of the gastric artery and ulcers. But the clinical history of most cases of ulcer of the stomach will not correspond with such a method of causation. A chronic obliterating endarteritis would seem to be a more probable cause.

Hæmorrhagic Erosions occur as rounded spots or narrow streaks, formed by a loss of substance of the mucous membrane. The mucous membrane at these points is congested, soft, and covered by small blood-clots. The destruction of the mucous membrane is usually superficial, but may involve its entire thickness. The number of these erosions may be so great that the entire internal surface of the stomach is studded with them. They give rise to repeated hæmorrhages, and are accompanied by catarrhal inflammation of the rest of the mucous membrane.

They occur at all periods of life, even in infants. Their usual seat is the pyloric portion of the stomach.

They may be idiopathic. Usually, however, they occur in connection with some serious general disease.

Follicular Ulcers somewhat resembling the ulcers of the small intestine are occasionally met with. They are produced by changes in the aggregations of lymphatic tissue, which are situated about the blind ends of the gastric tubules.

DILATATION.

Very considerable degrees of dilatation of the stomach are found at autopsies, without stenosis of the pylorus or any other mechanical cause to account for them. It is usually difficult to determine how long these dilatations have existed and how much effect they have in causing death. Nine such cases are recorded by Goodhardt.[1]

Acute Dilatation of the stomach, with vomiting of very large quantities of thin fluid, has been observed in a few cases.[2] It is a very curious condition, the dilatation of the stomach being developed suddenly and without discoverable cause.

Of the mechanical causes which produce dilatation of the stomach, stenosis of the pylorus is the most common. Such a stenosis may be effected by a tumor, by chronic inflammation and thickening, and by the cicatrization of ulcers. Less frequently obstructions of the small and large intestines act in the same way.

Some forms of chronic gastritis are attended with dilatation of the stomach without stenosis.

In rare cases, circumscribed, sacculated dilatations are produced by the presence of foreign bodies—portions of wood, metal, etc.

TUMORS.

Papilloma.—It has already been mentioned that in some cases of chronic gastritis there are small, polypoid hypertrophies of the mucous membrane. Besides these, we find polypoid tumors which may reach a considerable size. They are composed of a connective-tissue stroma arranged so as to form tufts covered with cylindrical epithelium. In some cases, there are also tubules lined with cylindrical epithelium, so that the tumor has partly the structure of an adenoma. *Fibromata* of small size are sometimes found in the connective-tissue coat. *Lipomata* are formed in the submucous connective tissue in the shape of rounded or polypoid tumors. They usually project inward, but sometimes outward beneath the peritoneum. They may also appear in the form of numerous yellow nodules beneath the mucous membrane.

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[1] Trans. Lond. Path. Soc., 1883, p. 88.

[2] Trans. Lond. Path. Soc., vol. iv. and vol. xxxiv., p. 82. *Hughes Bennett*, "Practice of Medicine." *Fagge*, Guy's Hospital Reports, vol. xviii., p. 1. *Andral*, Clinique Médicale.

Myomata occur in the form of rounded tumors which originate in the muscular coat, but may gradually separate themselves from it and project inward or outward. The submucous myomata are at first small tumors lying loosely attached in the submucous tissue. As they grow larger they push the mucous membrane inward and take the shape of polypoid tumors. *Lymphomata* in the wall of the stomach are seen in some cases of leukæmia.

Sarcomata are said to occur in the wall of the stomach in rare instances. It must be admitted that in some of the tumors of the wall of the stomach, which are ordinarily called cancerous, the structure is not well defined, and it is possible that some of them are sarcomata.

A myo-sarcoma growing outward from the greater curvature of the stomach is described by Brodowski.[1] The tumor weighed twelve pounds. It was composed largely of smooth muscle cells. There was a secondary tumor in the liver.

Adenoma.—It has been already mentioned that in some of the papillary tumors of the mucous membrane there is a considerable growth of tubules lined with cylindrical epithelium.

Besides these, we find in the submucous coat circumscribed tumors composed of tubules like those of the gastric mucous membrane.

Small tumors resembling the pancreas have also been seen in the submucous and subserous coats.

Carcinoma of the stomach is almost always primary. But very few secondary cases have been recorded.[2]

Primary carcinoma of the stomach is of the colloid variety, or common cancer, or cancer with cylindrical epithelial cells, or it is pigmented.

Colloid cancer is composed of a connective-tissue stroma, arranged so as to form cavities of different sizes, which contain colloid matter and polygonal cells. It infiltrates first the submucous connective tissue and then extends inward and outward. In this way there is formed a diffuse thickening of the pyloric end of the stomach rather than a circumscribed tumor. Sometimes the whole of the wall of the stomach is changed in this way. Secondary tumors are usually situated in the peritoneum.

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[1] Virch. Arch., Bd. 67, p. 227.

[2] Virch. Arch., Bd. 38 and 86, p. 159. Trans. Path. Soc., London, 1876, p. 264

Carcinoma with cylindrical epithelial cells. These tumors are formed of a connective-tissue stroma, which may contain numerous round cells, and of tubules lined with cylindrical epithelium like that of the mucous membrane of the stomach. In these tumors, the new growth seems to begin in the gastric tubules. As the arrangement of the tubules is more or less regular, these tumors may be called adenomata or carcinomata (see Fig. 69).

Common cancer is formed of a connective-tissue stroma inclosing rounded and tubular spaces filled with small, polygonal nucleated cells. In some cases this structure is well marked; in others, the stroma is abundant and filled with round cells, the spaces are very small, and the epithelial cells few. It may then be difficult to distinguish between inflammatory thickening, sarcoma, and carcinoma.

Both these forms of carcinoma, common cancer and cancer with cylindrical cells, run the same course as regards their gross appearance, their situation, and their development of metastatic tumors.

About sixty per cent of these tumors are situated at the pyloric end of the stomach, on the lesser curvature or on the posterior wall. The cardiac end of the stomach, the greater curvature, or nearly the entire wall of the stomach may also be the seats of the new growth, but not as frequently.

The new growth usually follows one or other of three types:

1. There is a circumscribed, flat tumor formed in the deeper layers of the mucous membrane and pushing this membrane inward. After a time, the mucous membrane over the centre of the tumor dies, the destructive process involves the tumor also, and so an ulcer with thickened edges is formed. In some cases the new growth extends laterally and outward, while the central destruction still continues; then the ulcers reach a large size, their walls and floor are thick, and peritoneal adhesions are formed over them. In other cases the ulcer perforates completely through the wall of the stomach, unless the opening is closed by adhesions to the neighboring viscera.

2. Large rounded tumors are formed, often several inches in diameter, which project into the cavity of the stomach.

3. There is a diffuse, flat infiltration of the deep layers of the mucous coat, of the connective-tissue coat, and sometimes of the muscular coat, which does not ulcerate and hardly forms a tumor. This infiltration may be confined to the pyloric end of the stomach, or may involve nearly the whole of its wall.

There is in most of the cases a good deal of chronic catarrhal inflammation of the mucous membrane.

If the pylorus is obstructed, the stomach is often dilated.

The new growth may extend from the stomach to the œsophagus, but it very seldom involves the duodenum.

Metastatic tumors are very common. The liver, the lymphatic glands, and the peritoneum are the parts most frequently affected, but such metastases have been seen in nearly every part of the body.

DEGENERATIONS.

Calcification of the mucous membrane of the stomach sometimes occurs as a metastatic process in connection with extensive diseases of the bones.

Waxy Degeneration sometimes involves the blood-vessels of the mucous membrane.

THE INTESTINES.

MALFORMATIONS.

Diverticula of the intestines occur in several different ways:

1. The abdominal walls are cleft asunder at the navel. The ileum opens through this cleft by a narrow aperture in its wall. The lower portions of the ileum and the colon are small or entirely closed.

2. There is an opening in the abdominal wall as before, but there is not a direct opening into the ileum. There is a long diverticulum of the ileum, with an open end projecting into the opening in the abdominal wall.

3. The abdominal wall is closed. There is a diverticulum of the ileum connected with the navel by a solid cord.

4. There is an unattached diverticulum of the intestine. This is much the most common form. The diverticula occur only in the lower part of the ileum. They usually spring from the convex surface of the intestine, more rarely from its attached border. In the latter case, they are joined to the mesentery by a fold of peritoneum. The diverticulum forms a pouch, one to six inches long, of about the same diameter as the intestine, smallest at its free extremity.

Such diverticula do not interfere with the functions of the intestines. They sometimes form part of a hernia. Sometimes the remains of these intestinal diverticula—called Meckel's diverticula—form soft, projecting tumors at the umbilicus in children. Microscopical examination of such tumors often shows the structure of the intestinal mucosa and muscularis. If they remain attached by a fibrous cord to the navel, this cord may be the cause of incarceration of a portion of the intestines.

Cloacæ consist in the union of the rectum, bladder, and organs of generation in a common outlet.

1. *Simple Cloacæ* are: (*a*) Complete, and consist in the common opening of the urethra or ureters, the vagina, and the rectum into the closed bladder, or into a sinus opening outward, which represents either the vagina or the rectum. (*b*) Incomplete. The rectum opens into the vagina, the bladder, or the urethra, while the lower part of the rectum is closed or absent.

2. *Cloacæ combined with Cleft Bladder.*—(*a*) The simple cleavage of the intestines is combined with cleft bladder. The anterior abdominal wall from the umbilicus to the symphysis, the symphysis, and the anterior wall of the bladder are absent; the gap is filled with a membrane which represents the posterior wall of the bladder. On to this membrane open the ileum, ureters, and vagina. (*b*) The intestine is perfectly formed, but the rectum opens into a common sinus with the ureters and vagina; or the ureters open into the cleft bladder, and the rectum and external genitals are united; or the ureters open into the rectum, and the latter terminates normally.

3. *Cloacæ combined with Abdominal Hernia.*—There is a hernial sac containing all the abdominal viscera. At the lower end of the sac is an opening leading into a sinus in which open the lower end of the ileum, the bladder or urethra, and the ureters. The rectum is absent.

Atresia Ani consists in a deficient development of the colon or rectum. The entire colon may be absent; the rectum may be absent, or represented by a solid cord; or the upper or lower part of the colon may be absent, or separated by a solid cord.

More rarely, blind terminations of the small intestines are found, and sometimes a narrowing so complete as to close the canal.

The intestines are also found abnormally shortened in various degrees.

INCARCERATION.

1. The most common form is that in which a portion of intestine is strangulated by a fibrous band. Such fibrous bands are produced by peritonitis, or are remains of fœtal growth. They pass from the intestines to the abdominal wall, or from one part of the intestines to another. The intestine becomes in some way caught under one of these bands, and is compressed by it. The stricture thus produced may cause a gradual accumulation of fæces in the intestine above it, and may last for a long time before death ensues. In other cases, the stricture interferes at once with the circulation of the blood; the intestine is intensely congested, becomes gangrenous, and death takes place with the symptoms of general peritonitis.

2. A portion of intestine becomes caught in some abnormal open-

ing in the mesentery or omentum, or in the foramen of Winslow, or between the two layers of the mesentery. We have seen a case in which twelve feet of intestine had passed through a small opening in the mesentery.

3. A coil of intestine makes half a turn at its base, so that the two sides of the loops cross at its base. In this way, the lumen of the intestine is completely closed, and the vessels are compressed, so that congestion, peritonitis, and gangrene result. This form of incarceration is most frequent in the ascending colon. In the small intestine, it only occurs when the gut is fixed by old adhesions.

4. A portion of the intestine, with its mesentery, makes one or more complete turns on itself, closing the canal and compressing the vessels.

5. A portion of the intestine makes a half or entire turn about its long axis. This is very rare, and only occurs in the colon.

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6. The mesentery of a part of the intestine is long and loose, in consequence of a dragging down of the intestine by a hernia or by habitual constipation. The portion of intestine thus permitted to hang down is habitually filled with fæces, and by its pressure on some other part of the intestine produces an incomplete stricture.

INTUSSUSCEPTION.

This change of position consists in the invagination of one portion of intestine in another portion. Usually this takes place in the direction of the peristaltic movements, from above downward; more rarely in the opposite direction.

The parts are found in the following condition: There are three portions of intestine, one within the other. The inner portion is continuous with the intestines above the intussusception; its peritoneal coat faces outward. The outer portion is continuous with the intestine below; its peritoneal coat also faces outward. The inner portion is turned inside out, its mucous membrane is in contact with the mucous membrane of the outer portion. In rare cases, the intussusception is complicated by the invagination of a second portion of intestine in the inner tube, and even by a third intussusception into the second one. These changes occur both in the large and small intestine; most frequently the lower part of the ileum is invaginated in the colon. The invaginated portion may be from a few inches to several feet in length. The lesion is most frequently found in early childhood.

The intussusception, by the dragging and folding of the mesentery which it produces, causes an intense congestion of the parts, and even large hæmorrhages between the coats of the intestine. The congestion may induce fatal peritonitis, or gangrene of the intestine, or chronic inflammation and adhesions, and the patient lives for a considerable time with symptoms of stricture. In other cases, the invaginated portion of intestine sloughs, the outer and inner portions become adherent, and the patient recovers, with or without some degree of stricture.

Besides this grave form of intussusception, we often find, especially in children, one or more small invaginations not attended with congestion or inflammation. These are formed during the death agony, or immediately after death.

TRANSPOSITION.

The position of the intestines may be the opposite to that which is usually found. The transposition may affect all the abdominal viscera, or only a single viscus is transposed.

WOUNDS—RUPTURES.

Penetrating wounds of the intestine usually prove rapidly fatal, either from shock or from peritonitis. Sometimes, however, the wound becomes closed by the formation of adhesions with the neighboring parts. Sometimes the wound in the intestines becomes adherent at the position of the wound in the abdominal wall, and an intestinal fistula is formed.

Rupture of the small intestine is not infrequently produced by severe blows on the anterior abdominal wall. It is noticeable that such blows may not produce any marks or ecchymoses of the skin. Such ruptures usually prove fatal very soon, but sometimes the patient lives several days, and the edges of the rupture undergo inflammatory changes.

Strictures of the intestine are sometimes followed by rupture of the dilated intestine at some point above the stricture.

THE SMALL INTESTINE.

INFLAMMATION.

Acute Catarrhal Inflammation of the greater part of the small intestine is developed as part of the lesion of cholera morbus, and after the ingestion of irritant poisons.

Acute inflammation of the duodenum accompanies gastritis, and occurs as an idiopathic condition.

Acute inflammation of the ileum occurs as an idiopathic condition, and accompanies inflammation of the colon and of the solitary and agminated glands.

In many of these cases we infer the existence of the inflammation from the clinical symptoms.

After death, the most marked lesions are the increased production of mucus and the congestion. In very severe cases, the inflammation may extend to the peritoneal coat.

Chronic Catarrhal Inflammation of the small intestine accompanies heart disease, phthisis, emphysema, cirrhosis of the liver, and Bright's disease. The intestine is coated with an increased amount of mucus; it is often congested; there may be a general thickening of all its coats.

Croupous Inflammation is produced by irritant poisons; it is associated with croupous colitis, and it occurs as an idiopathic disease. The mucous membrane is coated with fibrin, its stroma is infiltrated with fibrin and pus, and this infiltration extends to the connective tissue, muscular and peritoneal coats.

Suppurative Inflammation of the submucous connective-tissue coat is said to occur in rare cases. It is usually metastatic. It takes the form of purulent foci of variable extent, which perforate either inward or outward.

THE SOLITARY AND AGMINATED GLANDS.

It is not uncommon to find in healthy adults who have died from accidental causes a considerable swelling of the solitary and agminated glands (lymph nodules) of the ileum, without any reason which we can discover to account for this swelling.

Extensive burns of the skin may be followed by a very marked swelling of the solitary and agminated glands.

In persons who have died from the infectious diseases, it is not uncommon to find these glands swollen.

In children, swelling of these glands, often followed by softening and the formation of ulcers, accompanies many of the catarrhal inflammations of the large and small intestines.

In pulmonary phthisis, we very frequently find changes in the solitary and agminated glands of the small intestine, less frequently in the solitary glands of the colon. The changes seem to be of the same character as those which take place in tubercular inflammation of lymphatic glands in other parts of the body.

The glands become swollen, their elements are multiplied, tubercle granula are formed, the central portions of the glands become cheesy. The cheesy degeneration extends; it is followed by softening and by death of the mucous membrane over the glands; the softened tissue is discharged into the intestine, and ulcers are formed with overhanging edges. After this, the ulcer shows no tendency to heal, but, on the contrary, becomes larger, usually extending laterally so as sometimes to nearly encircle the gut. After death, we find, in different patients, these ulcers in all their stages of development. They vary much as to the proportion between the tubercular and the ordinary inflammatory changes. In some the tubercle granula are numerous, in others they are few or even absent altogether. The tubercle bacilli are very constantly found in them. There is also usually a tubercular inflammation of the peritoneum over the ulcers, and sometimes of the lymphatics and glands of the mesentery. Although these ulcers often reach a large size, it is but very seldom that they perforate into the peritoneal cavity.

Ulcers of the Duodenum.—A few cases have been recorded in which extensive burns of the skin have been followed within a few days by the formation of deep ulcers of the duodenum. It is still uncertain how these ulcers are produced.

Chronic perforating ulcers, resembling the chronic ulcers of the stomach, are found in the duodenum. They are associated with similar ulcers in the stomach or occur by themselves.

Some curious ulcers of the upper part of the small intestine are described by Israel.[1] There were five ulcers, from two and one-half to ten centimetres long, encircling the intestine, with irregular, granulating surfaces.

Syphilitic ulcers produced by changes in the solitary and agminated glands of the small intestine are sometimes found in infants.

EMBOLI.

Emboli have been found in the superior mesenteric artery in a number of cases; in the inferior mesenteric artery they are less frequent. They produce an intense venous congestion of the entire wall of the intestine, with hæmorrhage into its cavity and its wall.

THE LARGE INTESTINE.

INFLAMMATION.

The mucous membrane of the large intestine is very frequently the seat of acute and chronic inflammatory processes. The larger number of these belong to the condition which is described clinically under the name of dysentery. The inflammation affects most frequently the rectum, sometimes the entire length of the colon, sometimes only the upper part of the colon.

Acute Catarrhal Colitis.—The mucous membrane is swollen and congested, there is an increased production of mucus, there may be a large transudation of serum, there may be some bleeding from the capillary vessels. The epithelium desquamates, and the connective tissue and glandular coats are infiltrated with a moderate number of pus cells.

There are curious cases of catarrhal colitis occurring in old and feeble persons which are very fatal. After death, we find the mucous membrane soft and black, and the entire wall of the gut soft and easily torn. The intestine looks almost gangrenous, but yet does not seem really to be so. What the real nature of these cases is I do not know, although I have seen several of them.

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[1] Charité-Annalen, 1884, p. 707.

Chronic Catarrhal Colitis may succeed acute colitis, or it may begin as a chronic affection. Either a portion or the entire length of the colon may be involved, and the lesion is found developed in different degrees in different cases. The glandular coat is thickened by the growth of connective tissue between the tubules, or the tubules may become atrophied and the glandular coat is then thin. There may be a complete destruction of the glandular coat at different points, so that ulcers of different sizes are formed. The solitary follicles in the wall of the intestine may be inflamed and softened, and in this way also small round ulcers are formed. The connective-tissue coat is thickened; this thickening is uniform, or it is more marked at some places than others, so that little polypoid tumors are formed, which project into the cavity of the colon. The muscular and peritoneal coats are also thickened.

Croupous Colitis.—This form of inflammation may involve the rectum alone, or the entire length of the colon, or only its upper portion. The mucous membrane is congested and swollen, and coated with a layer of false membrane; the connective tissue between and beneath the glandular tubules is infiltrated with fibrin and pus, and in severe cases the inflammation involves the muscular and peritoneal coats also. The inflammation is usually more intense at some places than at others, so that the surface of the mucous membrane shows the false membrane in isolated patches. Less frequently there is a uniform coating with the false membrane. In mild cases, as the inflammation subsides, the products of inflammation are absorbed, and the wall of the intestine returns to its normal condition. In more severe cases, the quantity of the inflammatory products is so great that portions of the wall of the intestine become necrotic. This necrosis may involve only the glandular coat, or it may extend deeper into the wall of the intestine. The necrosed tissue after a time sloughs away, leaving behind ulcers of different sizes and depths. After this the ulcers may cicatrize, or their floors and walls may remain in the condition of granulation tissue for an indefinite length of time. When the latter is the case, there is added a chronic inflammation of the wall of the intestine between the ulcers, with changes in the mucous membrane and thickening of the connective tissue and muscular coats.

Small multiple *diverticula* are not infrequently found along the free border of the large intestine, which may be empty or contain fæcal matter. They appear to be formed, as are the multiple diverticula of the bladder (see Bladder), by the crowding of pockets of the mucous membrane through between the fibres of the muscularis. They appear to be usually of no special pathological significance, but they may become the seat of inflammation.

The Solitary Follicles.—In children, the lymphatic glands (lymph nodules) in the wall of the large intestine frequently become swollen, soften, and form ulcers in cases of catarrhal colitis. In adults, such changes are not common except in the dysentery of armies. In army dysentery, the changes in the solitary follicles often form the principal part of the lesion, although catarrhal or croupous inflammations are usually associated with them. The intestine is thickly studded with small, round ulcers originating in the destructive changes in the solitary follicles. But these ulcers show a disposition to increase in size and to remain as chronic ulcers for a long time.

The Cæcum.—Catarrhal inflammation of the cæcum is not uncommon. It is usually produced by an habitual accumulation of fæces in this part of the intestine. The course of the inflammation is chronic, but marked by acute exacerbations. At first the mucous membrane undergoes the ordinary changes of chronic catarrhal inflammation; then there is a slow suppurative inflammation which extends through the wall of the intestine, and produces ulcers and perforations. Through these perforations the fæces may pass into the peritoneal cavity, or the perforations are partly closed by adhesions, and abscesses are formed, or sinuses into the surrounding soft parts.

The Rectum.—Besides the inflammatory changes already described as existing in the colon, we sometimes find a suppurative inflammation of the connective tissue which surrounds the rectum, either associated with lesions of the mucous membrane or occurring by itself.

In adults, the lower end of the rectum is the part of the intestine which is the most frequent seat of syphilitic ulceration. Most of these ulcers seem to be the result of unnatural coitus, or of infection from specific sores of the vulva; but some of them seem to be due to the softening of gummy tumors.

The Vermiform Appendix.—The most frequent form of inflammation of the appendix is a suppurative one. The appendix is swollen and congested; its walls are infiltrated with pus; at some points there may be necrosis and sloughing of portions of its wall. Within the cavity of the appendix we find fæcal concretions, or foreign bodies, or nothing. Such an inflammation may terminate in resolution, but more frequently it sets up an inflammation of the surrounding tissues. This inflammation may be either a local or general peritonitis, or a suppurative inflammation of the soft parts about the appendix.

Less frequently there is a chronic inflammation of the mucous membrane of the appendix, followed by constriction of its upper portion, while the lower part is dilated into a cyst filled with mucus and serum. Tubercular inflammation of the appendix is of occasional occurrence.

TUMORS.

Myoma.—Tumors composed of smooth muscle and connective tissue grow in the muscular coat and project inward. They may be large enough to obstruct the intestine, and may then give rise to intussusception. In the duodenum, such tumors may obstruct the common bile duct. Less frequently, these tumors project outward into the peritoneal cavity.

Lipomata may be developed from the submucous coat and grow inward, or from the subserous coat and project outward into the peritoneal cavity.

Polypoid Tumors, projecting into the cavity of the intestine and composed of connective tissue and covered with epithelium, are frequently found. They are associated with catarrhal inflammation or occur by themselves. They are found throughout the intestinal tract, and may be single or multiple. They grow from the submucous coat, and project inward. Some of them are small, solid, connective-tissue tumors, covered by the mucous membrane which they have pushed inward. Others are of the same character, but of large size. In others, the connective tissue is arranged in branching tufts, covered with cylindrical epithelium, and in these last tumors there may also be tubules lined with cylindrical epithelium, giving to the growth the characters of an adenoma.

Adenomata are found in the duodenum and colon. They form flat infiltrations of the wall of the intestine, or project inward as polypoid tumors. They are composed of tubular follicles, like those of the intestinal mucous membrane, and of a connective-tissue stroma. In some of these tumors the tubules have a tolerably regular shape and arrangement; there is no infiltration of surrounding tissue; the tumor is of benign nature. In other tumors, the tubules are irregular in shape and arrangement, and the growth infiltrates the surrounding parts. There is no sharp dividing line between these tumors and the carcinomata.

Carcinomata are found in the colon and the duodenum, and are of three varieties.

1. The new growth is composed of tubules lined with cylindrical epithelium. It begins as a flat infiltration of the submucous coat, which soon surrounds the intestine, infiltrates the whole thickness of the wall of the gut, and may extend to the surrounding soft parts. Fungous masses project into the cavity of the intestine, while at the same time ulcerative and destructive processes are going on. According to the exact arrangement of the growth, there is more or less stenosis of the intestine.

2. The growth has the characters of colloid cancer, and forms a diffuse infiltration of the intestinal wall, completely surrounding it and often extending over a length of several inches.

3. At the anus there is sometimes a carcinomatous growth, with epithelial cells (epithelioma), like similar growths in the skin, which volves the lower end of the rectum.

Lymphoma.—Tumors composed of tissue resembling that of lymphatic glands originate in the solitary and agminated follicles and the intestinal wall in cases of leukæmia and pseudo-leukæmia.

Similar tumors are found as an idiopathic lesion both in the la and small intestines. These tumors are irregular diffuse growths filtrating the wall of the intestine, the mesentery, and the neighbor glands, and reaching a considerable size. They often ulcerate interna and produce dilatation or stenosis of the intestine. It is hard to whether some of these tumors should be called lymphomata or sar mata.

CONCRETIONS.

There are sometimes found in the intestines round, oval, or irregu masses of firm consistence. They are usually small, but may reach t size of a man's fist. They are composed of fæcal matter, mucus, bi the carbonate and phosphate of lime, and triple phosphate. They m produce inflammation, ulceration, and perforation.

PARASITES.

Mycosis intestinalis.—Under this name have been described number of cases in which inflammation of the intestines occurred one of a group of lesions.[1] In these cases, there are found ecchymos of the skin and mucous membranes; patches of fibrin in the stomac small intestine, and colon; serum in the peritoneal cavity; swelling the spleen and lymphatic glands; and sometimes inflammation of t lungs. The intestinal lesions are most marked in the small intesti and the upper part of the colon. The mucous membrane is studd with small brownish patches. At the centres of these patches, the wa of the intestine is infiltrated with pus, and around the centres it is infi trated with blood. Bacilli resembling those of anthrax are found in t intestinal lesions and in other parts of the body. It is believed that t disease is a form of anthrax.

Ascaris lumbricoides is found in the small intestine, either singly in considerable numbers. In rare cases, a number of worms may for a mass which produces inflammation, ulceration, and perforation.

Oxyuris vermicularis is found in large numbers in the rectum.

Tricocephalus dispar is found in the cæcum.

Ankylostomum duodenale is found in the duodenum, and may giv rise to considerable hæmorrhages.

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[1] Virch. Arch., Bd. xxi., p. 579; xxx., p. 366; lii., p. 541. Zeitsch. f. Biologie v., 129. Arch. d. Heilkunde, xv. Arch. f. klin. Med., xii., p. 517.

Trichina spiralis is found in its adult condition in the small intestine.

Pentastomum denticulatum occurs in the submucous tissue of the small intestine in an incapsulated condition.

Cysticercus cellulosæ has been seen, in a few cases, on the mucous membrane.

Tænia solium, Tænia mediocanellata, and *Bothriocephalus latus* are all found in the small intestine.

Very large numbers of various forms of bacteria are regularly found in the intestinal cavity intermingled with its contents and clinging to its walls.

THE PERITONEUM.

The free surface of the parietal peritoneum is covered with a single layer of flat, polygonal nucleated cells. Beneath these cells are successive planes of connective tissue extending down to the muscles and fasciæ. These planes are formed of a fibrillated basement substance, reinforced by elastic fibres, and of branching cells. Imbedded in the connective tissue are the nerves, blood-vessels, and lymphatics. The lymphatic system is very extensive.

The omentum consists of fibrillated connective tissue arranged so as to form a meshwork. The trabeculæ of the meshwork are completely covered by large, flat cells. In the basement substance, beneath the endothelium, are branching cells. In the larger trabeculæ are blood-vessels, lymphatics, and fat. Sometimes we find on the larger trabeculæ little nodules formed of polygonal or branched cells.

MALFORMATIONS.

Arrest of development of the peritoneum occurs in the shape of fissures in the mesial line or external to it; in the case of the diaphragm being absent, of a fusion with the pleura; and as defective development of the mesentery, the omentum, and the other folds of the peritoneum.

Excess of development occurs in the shape of unusual length of the mesentery, the omentum, and the other folds of the peritoneum; or of supernumerary folds and pouches. These are chiefly found in the hypogastric, iliac, and inguinal regions and near the fundus of the bladder. There is access to these sacs by a well-defined fissure or ring, which is frequently surrounded by a tendinous band lying in the duplicature. They may give rise to internal incarceration of the intestines.

INFLAMMATION.

The very great extent of the peritoneum, and the readiness with which its lymphatic system absorbs foreign matters from the peritoneal

cavity, render peritonitis a most severe and dangerous form of inflam
tion.

If the greater part of the peritoneum is inflamed, we call the lesi
general peritonitis. If only a circumscribed area is involved, it is a
peritonitis. The course of the inflammation may be rapid or slow
that we speak of *acute* and *chronic* inflammation. The inflamma
may be attended with the production of tubercle tissue, and then it
tubercular peritonitis.

I. Acute Peritonitis.

The acute inflammations of the peritoneum may occur as idiopat
lesions without discoverable cause; but much more frequently they
directly due to some appreciable cause.

Wounds and contusions of the wall of the abdomen; wounds, ulc
new growths, incarcerations, intussusceptions, ruptures, perforati
and inflammations of the stomach and intestines; inflammation of
vermiform appendix; injuries, ruptures, and inflammations of
uterus, ovaries, and Fallopian tubes; rupture and inflammation of
bladder; inflammation of and about the kidneys; abscesses and hyda
cysts of the liver; inflammation of the gall bladder and large
ducts; thrombosis of the portal vein; inflammations of the spleen, p
creas, lymphatic glands, retroperitoneal connective tissue, verteb
ribs, and pelvic bones; septicæmia and the infectious diseases, a
chronic Bright's disease—are all ordinary causes of acute peritonitis.

According to the exact cause of the inflammation, the peritonitis is
first either local or general. A local peritonitis may remain circu
scribed, or it may spread and become general.

We can distinguish two anatomical forms of acute peritonitis.

1. *Cellular Peritonitis.*—This form of peritonitis may be produc
by any irritant which does not act too energetically. It can be excit
in dogs by injections of very small quantities of a solution of chloride
zinc. In the human subject we find it with perityphlitis, with circu
scribed abscesses in the peritoneal cavity, and in cases of puerperal fev
which die within forty-eight hours after the development of symptom

After death we find the entire peritoneum of a bright-red colo
from the congestion of the blood-vessels; but there are no fibrin,
serum, no pus, no other lesions visible to the naked eye. Minute exa
ination, however, shows a very marked change in the endothelial cell
They are increased in size and number, and the new cells coat the su
face of the peritoneum and project outward in little masses (Fi
156).

2. *Exudative Peritonitis.*—The ordinary form of acute peritonit
is attended with the production of serum, fibrin, and pus, and wit
changes in the endothelium and connective-tissue cells.

If we inject a solution of chloride of zinc or of some other irritant into the peritoneal cavity of a dog, we find that by the end of one or two hours inflammatory changes are evident. There is a little serum in the peritoneal cavity, a general congestion of the peritoneum, and little knobs and threads of fibrin on its surface. There are no marked changes in the endothelium or connective-tissue cells, but pus cells are present in moderate numbers in the stroma, just beneath the endothelium, and white blood-cells in the vessels.

After the lapse of twenty-four hours, the lesions are more marked. The congestion of the peritoneum is much more decided, there is more serum in its cavity and a thicker layer of fibrin and pus on its surface. Minute examination shows that two distinct sets of changes are going on at the same time: (1) a production of fibrin, serum, and pus; (2) a swelling and multiplication of the endothelial cells. If the inflammation is very intense, the pus and fibrin are most abundant; if the inflammation is milder, the changes in the endothelium are more marked. The fibrin coagulates on the free surface of the peritoneum. The white blood-cells collect in large numbers in the blood-vessels, and as pus cells infiltrate the stroma and collect on its surface. There is no special

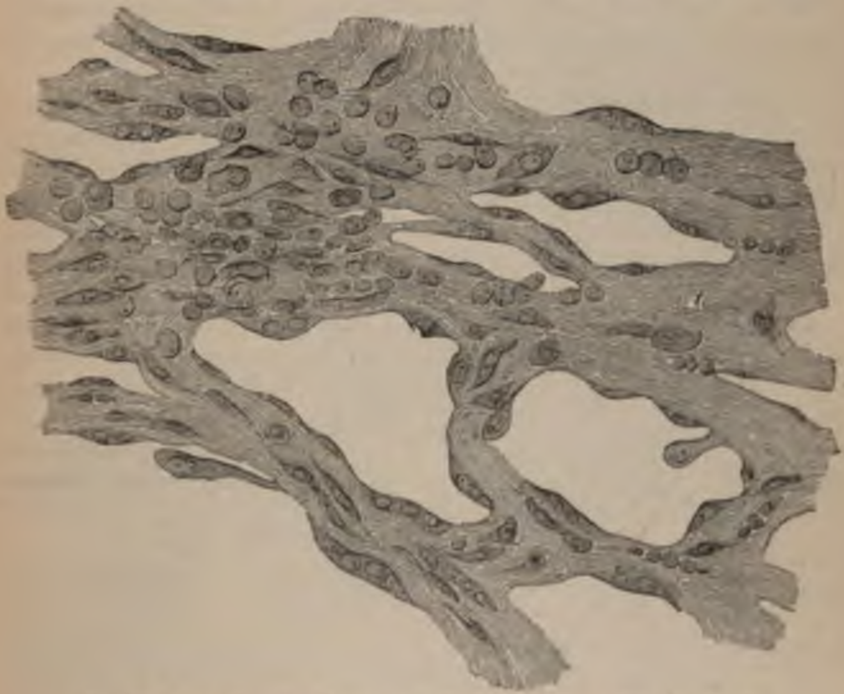

FIG. 156.—ACUTE CELLULAR PERITONITIS—Human omentum, × 750 and reduced.

change in the connective-tissue cells. The endothelial cells may ren
in place, although their edges and corners are separated by pus cells
knobs of fibrin; or the endothelium falls off in large patches; or
surface of the peritoneum is covered with numerous cells which look
endothelial cells more or less deformed. But few dogs survive the th
day of an acute artificial peritonitis.

In the human subject, if death takes place before the third day, b
the gross and minute changes are the same as those seen in the d
There are present the same general congestion, the pus, fibrin,

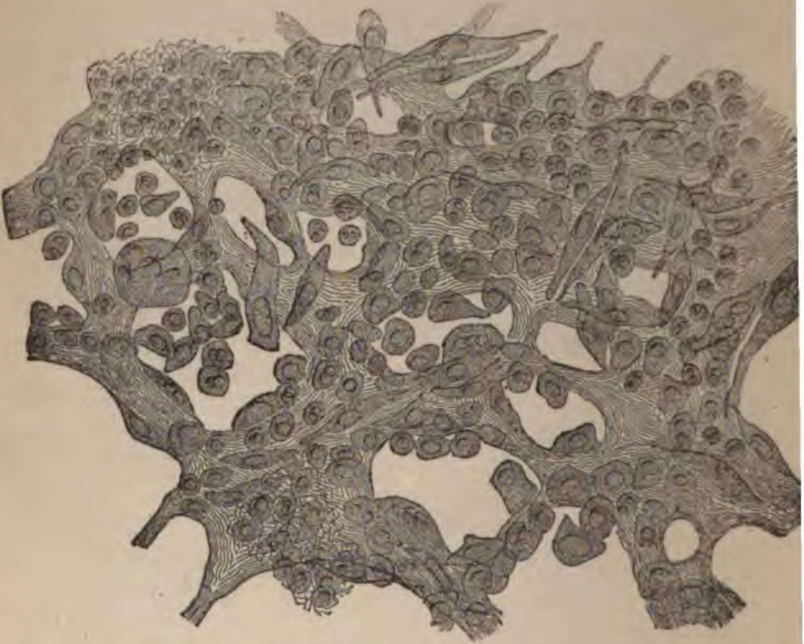

FIG. 157.—ACUTE EXUDATIVE PERITONITIS of eight days' duration—Human omentum, × 850 a
reduced.

serum, the desquamation and multiplication of the endothelial ce
(Fig. 157).

In many cases of peritonitis, however, death occurs between the six
and fourteenth days of the disease. The appearance of the peritone
at this period of the inflammation is not always the same. The cong
tion of the blood-vessels may persist, it may be very intense and acco
panied with extravasations of blood, or it may be entirely absent. The
may be a thin coating of fibrin and pus gluing together neighboring su
faces of peritoneum, or this layer may be very thick. The accumulati
of pus may be superficial, or it may infiltrate the whole thickness of t
peritoneum and the subperitoneal connective tissue. The quantity

purulent serum in the peritoneal cavity may be small or large, and this serum may contain few or many pus cells, or the serum may be of a dirty brown color and filled with bacteria. When the purulent serum is shut in by adhesion, it is often thick and yellow, like the pus of an abscess.

The minute appearances differ from those seen at an earlier stage, chiefly in the larger amount of inflammatory products, and in the changes in the fixed connective-tissue cells. During the first three days of an acute peritonitis, the connective-tissue cells are but little changed, but by the seventh day there is a marked increase in their size and number.

Acute peritonitis may prove fatal by the fourteenth day; or it may be succeeded by chronic peritonitis; or the patients recover and permanent connective-tissue adhesions and thickenings of the peritoneum are left behind. Recovery is most common when the peritonitis has been a local one.

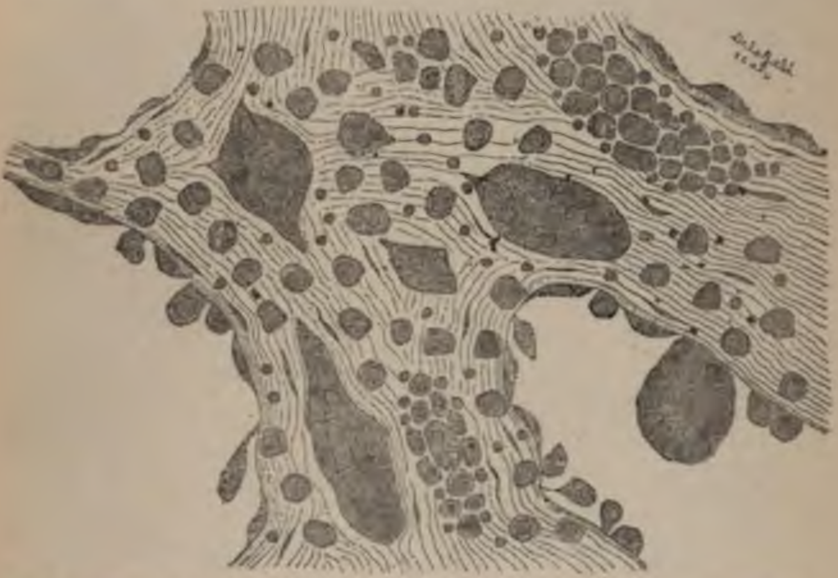

FIG. 158.—CHRONIC CELLULAR PERITONITIS occurring with pulmonary phthisis—Human omentum, × 750 and reduced.

II. *Chronic Peritonitis.*

We find the following varieties of chronic peritonitis:

1. *Cellular Peritonitis.*—This form of peritonitis is found as a complication of chronic endocarditis, of cirrhosis of the liver, of chronic pulmonary phthisis, and of acute general tuberculosis.

Neither fibrin nor pus is present, but there may be clear serum in the peritoneal cavity. The peritoneum may look normal to the na eye, or it may be studded with very minute, translucent nodules.

Minute examination shows changes in the endothelial cells and connective-tissue cells. These cells are everywhere increased in num and altered in shape; or, to speak more guardedly, the surface of peritoneum is covered with cells which look as if they were derived fr the endothelium and the connective-tissue cells (Fig. 159). Some large, flat cells; some smaller, polygonal cells; some irregularly fusifo some large, granular masses containing a number of nuclei. Altho

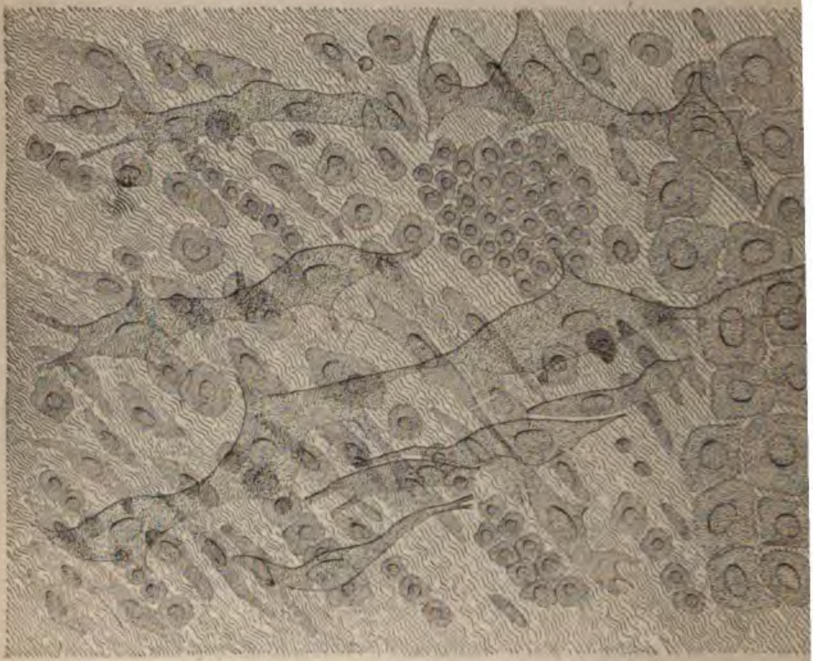

FIG. 159.—CHRONIC PERITONITIS WITH ADHESIONS, × 750 and reduced. Parietal peritoneum.

these new cells are found over most of the surface of the peritoneu yet they are more numerous in little patches which are scattered he and there.

2. *Peritonitis with Adhesions.*—There may be a formation of pe manent adhesions without the production of fibrin or pus. It is ofte indeed, difficult to tell whether old peritoneal adhesions are due to t form of chronic peritonitis of which we are now speaking, or wheth they are the result of an acute peritonitis. But there are some cas in which the mode of development of the adhesions seems evident.

If, from perityphlitis or some other cause, a collection of pus is shut in in some part of the peritoneal cavity, we may find the rest of the peritoneum smooth and shining; no serum, fibrin, or pus, no thickening; but the neighboring surfaces of the peritoneum are attached to each other by adhesions. These adhesions are in the shape of threads and membranes, often of the most extreme tenuity. They are formed of a fibrillated basement substance, the fibrils crossing each other in all directions. In the basement substance are cells, some fusiform and stellate, but most of them look like large branching cells, of which the cell bodies have become fused with the basement substance while the nuclei remain.

Close to these adhesions the peritoneum may appear normal to the naked eye, but if it is put in water very fine threads and membranes will float upward from its free surface. Minute examination shows that the connective-tissue cells are increased in size and number, that the endothelial cells are replaced by cells of a great variety of shapes, and that the thin little threads and membranes on the surface are formed of large branching cells (Fig. 159).

Such a peritonitis with adhesions appears to be a more advanced stage of the cellular peritonitis just described, but the inflammation, instead of stopping at the production of cells alone, goes on to the formation of membranes.

We sometimes find in the same patient chronic pleurisy with adhesions and chronic peritonitis with adhesions.

3. *Chronic Peritonitis with Thickening of the Peritoneum.*—This form of peritonitis occurs quite frequently as an idiopathic lesion. It may involve the greater part of the peritoneum or be confined to the capsules of the liver and spleen.

The most marked feature of the lesion is the thickening of the peritoneum—a thickening which may reach as much as an inch. The outer portions of the thickened peritoneum are composed of dense connective tissue, the inner layers of granulation tissue. The surface of the peritoneum is smooth or covered with fibrin. There may also be connective-tissue adhesions between different parts of the peritoneum. The peritoneal cavity contains clear or purulent serum.

In some cases the parietal peritoneum is principally involved; in others, the peritoneum of the stomach, intestines, liver, and spleen. The thickening of the capsule of the liver is attended with a diminution in the size of that viscus.

4. *Chronic Peritonitis with the Production of Fibrin, Serum, and Pus.*—This form of peritonitis may follow acute peritonitis, may be due to lesions of the abdominal viscera, or may occur without known cause.

The abdominal cavity contains purulent serum, either free or shut in by adhesions. The surface of the peritoneum is coated with fib and connective-tissue adhesions. The coils of intestine, and all neighboring surfaces of the peritoneum, are matted together partly fibrin, partly by permanent adhesions.

5. *Hæmorrhagic Peritonitis.*—This occurs most frequently as local inflammation. It involves the peritoneum behind and around uterus in the female, and that covering the recto-vesical excavation the male. The affected portion of the peritoneum is covered with lay of new membrane infiltrated with blood. The membranes are form of connective tissue containing numerous blood-vessels and infiltra with blood. The extravasations of blood may form tumors of consid able size.

General hæmorrhagic peritonitis is described by Friedreich.[1] In t cases of ascites, which had been frequently tapped, he found the visce and parietal peritoneum covered with a continuous membrane of a d fuse yellowish-brown color, mottled with extravasations of blood. T membrane was thickest over the anterior abdominal wall. It could separated into a number of layers. These layers were composed blood-vessels, masses of pigment, branching cells, and fibrillated ba ment substance. In many places the extravasated blood was coagulat in the shape of round, hard, black nodules. The entire new membra could be readily stripped off from the peritoneum.

6. *Tubercular Peritonitis.*—This occurs as one of the lesions acute general tuberculosis, with chronic pulmonary phthisis, wi tubercular inflammation of the genito-urinary tract, and as a lo inflammation.

The gross appearance of the lesion varies.

When tubercular peritonitis occurs as one of the lesions of gener tuberculosis, there are numerous small miliary tubercles, increase in t size and number of the endothelial and connective-tissue cells, and som times a little fibrin. Some of the miliary tubercles are composed tubercle tissue, others of round and polygonal cells.

As a complication of tuberculosis of the genito-urinary tract, we fi the peritoneum studded with miliary tubercles, coated with fibrin, a serum is also present in the peritoneal cavity.

As a complication of chronic phthisis, there are miliary tubercles the peritoneum of the small intestine immediately over tubercular ulce of the mucous membrane. There may also be thickening of the per toneum and permanent adhesions.

Local tubercular peritonitis usually follows one of three types:

(*a*) *Tubercular Ascites.*—The peritoneum is thickened; it is studde with masses of tubercle tissue in the form of miliary tubercles or large, flat masses. The omentum may be much thickened. There are but few adhesions, but there is a large amount of turbid serum. There may be at the same time tubercular pleurisy, or tubercles in the spleen or in the lymphatic glands (lymph nodes).

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[1] Virch. Arch., Bd. 58, p. 35.

(*b*) *Tubercular Peritonitis with the Production of a large amount of Fibrin.*—The peritoneum is studded with miliary tubercles. It is coated with a thick layer of soft, gelatinous fibrin, which mats together all the neighboring peritoneal surfaces, so that the abdominal cavity seems to be filled with a large, boggy mass composed of all the viscera adherent to each other, and with the interstices between them filled with fibrin.

(*c*) *Tubercular Peritonitis with Adhesions.*—The peritoneum is thickened and there are numerous connective-tissue adhesions. All the abdominal viscera are firmly matted together, and there may be collections of pus shut in by the adhesions. The adherent coils of intestine may ulcerate and open into each other. There are miliary tubercles, or large, tubercular nodules or plates.

TUMORS.

Fibromata are developed from the subperitoneal connective tissue, and project inward into the peritoneal cavity. They are found beneath the parietal peritoneum and that covering the intestines. Such tumors may reach a very considerable size.

Lipoma.—Circumscribed tumors composed of fat tissue are formed beneath the intestinal and parietal peritoneum. These tumors may become changed into fibrous tissue or calcified. Their pedicles may become atrophied so that they are left free in the peritoneal cavity.

When they grow beneath the parietal peritoneum, they may form fat herniæ. At the umbilicus, in the inguinal canal, along the vas deferens, in the crural ring, and in the foramen obturatorium, fatty tumors may grow, project outward under the skin like herniæ, and, by drawing the peritoneum after them into a pouch, may open the way for a future intestinal hernia.

Plexiform Angio-Sarcoma.—Very large tumors, resembling in their gross appearance colloid cancer, have been described by Waldeyer.[1] They are formed by a new growth of blood-vessels, with a production of gelatinous tissue from their adventitia.

Carcinoma of the peritoneum is either secondary or primary. The primary tumors assume the character of colloid cancer or of common cancer.

The colloid form frequently involves the greater part of the peritoneum, and forms a large mass which distends the abdomen. The omentum is changed into a large, gelatinous mass; the subjacent muscles, the lymphatic glands, and the liver are infiltrated with the new growth, a soft, gelatinous masses project into the peritoneal cavity. The umbi cus is sometimes invaded, so as to project outward in the form of a sem translucent tumor. The appearance of the new growth is that of a so jelly-like mass imbedded in a fibrous stroma. The minute structure that of a connective-tissue stroma, arranged so as to form cavities different sizes. These cavities are filled with a homogeneous, gelatino basement substance and with polygonal cells.

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[1] Virch. Arch., Bd. 55, p. 134.

Common carcinoma appears in the form of numerous small nodul scattered everywhere in the inner layers of the peritoneum. The

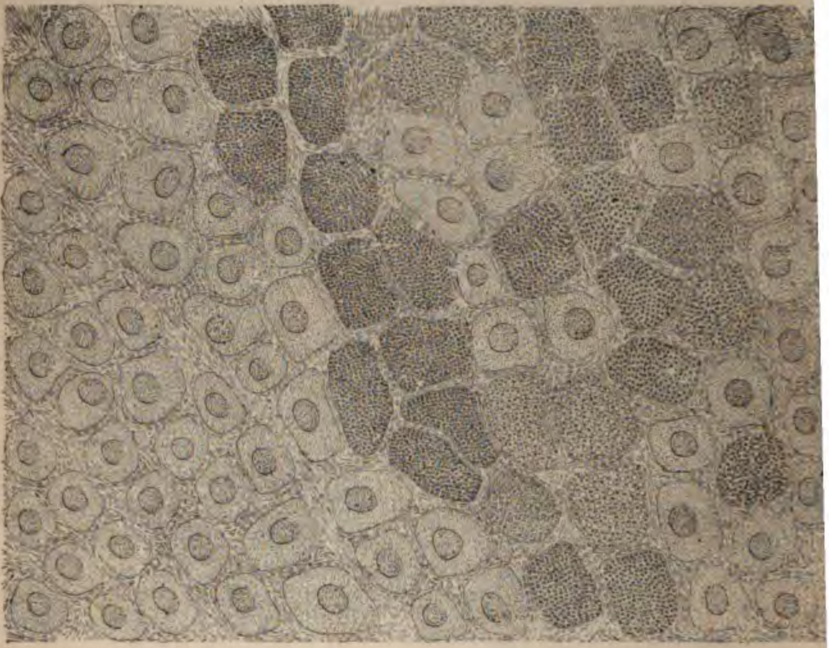

FIG. 160.—SECTION OF A RETROPERITONEAL SARCOMA, × 850 and reduced.

nodules are small, firm, and white, and are composed of a fibrous strom inclosing cavities filled with polygonal cells. With the formation o these nodules there are often associated a general thickening of the peritoneum, an accumulation of serum in the peritoneal cavity, and adhesions.

Endotheliomata of the peritoneum have been observed in cases wit similar growths in the pleura (page 226).

Sarcomata appear in the form of solitary, slowly growing tumor behind the peritoneum or between the folds of the mesentery.

These retroperitoneal sarcomata are found both in children and adults. They usually originate behind the peritoneum covering the posterior part of the abdominal wall. At first they grow slowly inward, pushing forward the peritoneum and abdominal viscera. After a time they assume a more infectious character, infiltrating the soft parts with which they come in contact, and forming metastatic tumors in the liver and other viscera.

These tumors are composed of a stroma and cells. The cells are large, of cuboidal shape, and often undergo fatty degeneration, when they become swollen. The proportion between the cells and the stroma and the arrangement of the cells vary in the different cases and in different parts of the same tumor (Fig. 160).

The stroma may be abundant and the cells scattered irregularly, each cell in a little cavity of its own. The stroma may be abundant, but the cells are collected in masses, as in a carcinoma.

The cells are abundant and close together, but each cell is surrounded by a thin partition of connective tissue. The blood-vessels are numerous, and the cells are arranged around them with some regularity.

From the above description, it will be seen that these tumors are of peculiar structure, and perhaps do not really belong to the sarcomata.

PARASITES.

Echinococci can be formed in their regular way at any part of the visceral and parietal peritoneum, or be free in the peritoneal cavity. These cysts may be small, or so large as nearly to fill the abdominal cavity.

Cysticercus cellulosæ may also be developed in the subperitoneal connective tissue.

MALFORMATIONS.

Congenital malformations of the liver are not common, and little practical importance. The organ may be entirely wanting; th may be diminished or increased in number; its form may be alte that it is rounded, flattened, triangular, or quadrangular. Th bladder or gall ducts may be wanting; the ductus choledochus n double, both ducts emptying into the duodenum, or one emptyin the duodenum, the other into the stomach. The single ductus dochus may also empty into the stomach. Owing to abnormal op in the diaphragm or the abdominal parietes, the liver may suff placement upward or forward. In congenital transposition of the vi the liver is found on the left side, the stomach and spleen on the side.

Small, isolated bodies, having the same structure as the liver been found in the suspensory ligament.

ACQUIRED CHANGES IN SIZE AND POSITION.

As a result of tight lacing, very marked changes are sometime duced in the shape of the liver. By the narrowing of the base thorax, the organ is compressed from side to side, and its convex s is pressed against the ribs. In consequence of this there are ridges and furrows on its convex surface. In consequence also circular constriction, a part of the right, and usually of the left lob fatty and amyloid degeneration, by congestion, and sometimes by cirrhosis, etc.

It may be diminished in size by atrophy, by cirrhosis, by acute parenchymatous degeneration, etc.

Changes in the position of the liver are produced by alterations in its size, by pressure downward from the thoracic cavity and upward from the abdomen, by the constriction of tight lacing, by tumors or circumscribed serous exudation between the liver and diaphragm, by curvature of the spine.

The liver is readily turned, by pressure from above or below, on its transverse axis. The transverse colon may be fixed above the liver so as to push it backward, downward, and to the right. There are a few cases recorded of dislocated and movable livers. These occurred in women who had borne children and whose abdominal walls were lax. With ascites it is not uncommon to find the liver quite movable.

ANÆMIA AND HYPERÆMIA.

Anæmia of the liver may be general or partial. It may be due to general anæmia or to local disturbances of the circulation, such as swelling of the cells in parenchymatous or other degeneration, pressure of tumors, etc. The organ appears pale, often of slightly yellowish or brownish color. It may be harder than usual, and smaller.

Hyperæmia of the liver is either an active or a passive process. In health, the amount of blood in the liver varies at different times, being regularly increased during the process of digestion. When the digestive process is unduly influenced by the ingestion of spirits, spices, etc., the hyperæmia assumes abnormal proportions, and when this is often repeated it may lead to structural changes in the organ. Severe contusions over the region of the liver sometimes cause a hyperæmia, which may result in suppurative or in indurative inflammation. In hot climates and in malarious districts, active and chronic hyperæmia of the liver are frequent, and often cause structural lesions. In scurvy, also, the liver is sometimes congested. Cessation and suppression of the menses and hæmorrhoidal bleeding may cause hyperæmia of the liver. In all these varieties of active congestion, the liver is enlarged, of a deep-red color, and blood flows freely from its cut surface.

The passive congestions of the liver are produced by some obstruction to the current of blood in the hepatic veins. Valvular diseases of the heart, emphysema and fibrous induration of the lungs, large pleuritic effusions, intrathoracic tumors, angular curvature of the spine, aortic aneurisms pressing on the vena cava, and constrictions of the vena cava and of the hepatic veins, may all produce a chronic hyperæmia of the liver. In all these cases, as the congestion affects principally the hepatic veins, we find the centre of each acinus congested and red while its periphery is lighter colored. This gives to the liver a mottled or meg appearance (*nutmeg liver*). The liver cells in the centre of acinus are frequently colored by little granules of red or black pigm and the cells at the periphery become fatty, so that the nutmeg app ance is still more pronounced. A liver in this condition is usuall medium size, but may be smaller or larger than normal.

When the congestion is long-continued, the veins at the centr each acinus may become permanently dilated, the hepatic cells in t meshes become atrophied (Fig. 161), so that the centre of each aci

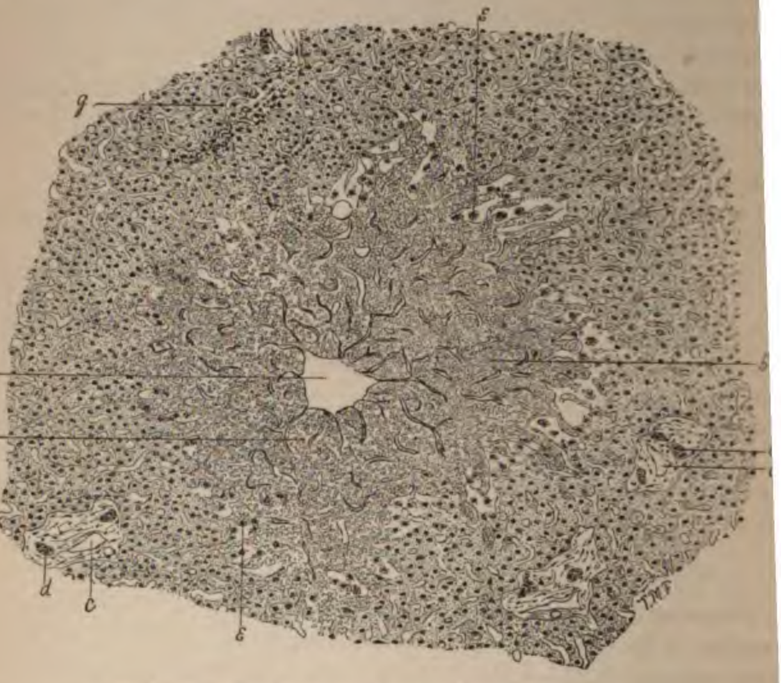

FIG. 161.—CHRONIC CONGESTION OF THE LIVER (Nutmeg liver).

This section shows complete atrophy of the liver cells at the centre of the lobule. *a*, dilat vena centralis; *b*, dilated capillaries filled with blood; *c*, portal vein surrounded by connective t sue; *d*, gall duct; *e*, atrophied liver cells; *g*, nearly normal liver tissue.

consists only of dilated capillaries or of these and new connective tissu or the dilatation and atrophy of the liver cells may, in circumscribe portions of the organ, involve the entire acinus. In long-continue congestion, the liver is usually smaller than normal, and may be slightl roughened or uneven on the surface; but it is sometimes enlarged. Th peculiar nutmeg appearance may be very well marked, or it may not b evident, the organ being of a dark-red color.

WOUNDS, RUPTURE, AND HÆMORRHAGE.

Wounds of the liver may induce hæmorrhage, which, if life continue, is followed by inflammation. Serious wounds of the liver are usually fatal, but recovery may occur even after the destruction of a considerable portion of the organ.

Rupture of the liver may be produced by severe direct contusions or by falls. It may be produced in children by artificial delivery. The rupture usually involves both the capsule and a more or less considerable portion of the liver tissue. It is commonly accompanied by large hæmorrhage, and is usually fatal.

Hæmorrhage.—Extravasations of blood in the substance of the liver, or more frequently beneath the capsule, are found in new-born children after tedious or forcible labors. In adults, hæmorrhage, except as the result of injury, is uncommon. Extravasations of blood are sometimes seen in malignant malarial fevers, especially in tropical climates; in scurvy, purpura, and phosphorus poisoning; and bleeding may occur in and about soft tumors, abscesses, and echinococcus cysts. It may also occur as a result of thrombosis of the hepatic vein.

LESIONS OF THE HEPATIC ARTERY.

The hepatic artery is in rare cases the seat of aneurisms which may attain a large size. Such aneurisms may displace the liver tissue, compress the bile ducts so as to cause jaundice, and may rupture into the stomach or abdomen.

Owing to its abundant anastomoses, emboli of the branches of the hepatic artery usually induce no marked lesions, but they sometimes result in hæmorrhagic infarctions.

LESIONS OF THE PORTAL VEIN.

Thrombosis, Embolism, and Inflammation.—*Thrombosis* of the branches of the portal vein may be produced by weakening of the circulation from general debility—*marasmatic thrombi* ; by pressure on the vessel from without, as in cirrhosis, tumors, gall stones, dilatation of the bile ducts, etc.; by injury; by the presence of foreign materials within the vessel; and as a result of inflammation of its wall, or of embolus. The thrombus may form in the vessels in the liver, or be propagated into them from without. It may partially or entirely occlude them. The clot may become organized as a result of endophlebitis, and a permanent occlusion of the vessel ensue. If the clot be a simple, non-irritating one, leading to occlusion, the consequences are usually more marked in the abdominal viscera than in the liver itself. The branches of the hepatic artery form sufficient anastomoses to nourish the liver tissue and prevent its necrosis, even in complete occlusion of the portal vein; and if occlusion occur slowly, the organ may continue to perform its functions.

But this obliterative form of thrombosis is usually attended by asci enlargement of the spleen, dilatation of the abdominal veins, and so times by hæmorrhage from the stomach and intestines.

In another class of cases, in addition to the local and mecha cal effects of a thrombus, there may be necrotic changes and suppurat inflammation in the walls of the vessels, or in the liver tissue about the The thrombi are apt to soften and break down, and the fragments n be disseminated through the smaller trunks of the portal vein. In t way, by the distribution through the smaller vessels of a disintegrat thrombus from a large trunk, or by the introduction into the branc of the portal vein of purulent or septic material from some of the dominal viscera or from wounds, multiple foci of purulent inflammati in the portal vein, and multiple abscesses involving the liver tissue, m be produced. In many cases, the presence of bacteria may be detect in the inflammatory foci.

These soft thrombi of the portal vein and the accompanying py phlebitis and abscess may be caused in a variety of ways. Ulcerati of the intestines and stomach, abscesses of the spleen, suppurative i flammation of the mesentery and mesenteric glands, inflammation a ulceration of the bile ducts from gall stones, inflammation of the umbi cal vein in infants, may all induce thrombi in their respective vein which may be propagated to the portal vein or may give rise to purule or septic emboli. Two cases are recorded in which a fish bone in t portal vein induced suppurative inflammation in that vessel. One these cases, occurring in Bellevue Hospital in 1867, was reported by D Janeway. Male, 47; dying, after a four weeks' illness, in a typhoid co dition, with lesions of sero-fibrinous peritonitis and chronic diffuse n phritis. There were numerous small abscesses in the right lobe of t liver, two in the left lobe. The left division of the portal vein contain a firm red and white clot over an inch long; the right division was line with a firm thrombus. The walls of the vein were thickened and contained purulent fluid. A fish bone, two inches long, its centre covere by a thrombus, lay half in the mesenteric and half in the portal vein.

In certain cases of thrombosis and inflammation of the portal vein the cause cannot be discovered.

In infants, inflammation of the umbilical vein may not only induc inflammation of the portal vein and abscesses in the liver, but multipl abscesses in various parts of the body, and acute peritonitis may be induced.

Rupture of the Portal Vein, with fatty degeneration of its walls, ha occurred in a few instances.

Chronic Endophlebitis, with atheroma and calcification, may occur i the walls of the portal vein, giving rise to thrombosis.

Dilatation of the Portal Vein, either uniform or varicose, may occu in various parts of the vessel or its branches. It may be caused by destruction of the liver capillaries in cirrhosis, or by occlusion of the vein by thrombi, tumors, etc.

THE HEPATIC VEINS.

The hepatic veins present lesions similar to those of the portal vein and its branches, but they are much less frequent. They may be dilated by obstruction to the passage of venous blood into the heart. They may be the seat of acute and chronic inflammation, and soft thrombi and suppurative inflammation may be produced by abscesses in the liver.

ATROPHY OF THE LIVER.

Atrophy of the liver may affect the entire organ or be confined to some part of it. General atrophy may occur in old age as a senile change, or may be induced by starvation or chronic exhausting diseases. The organ is diminished in size, is usually firm, and the acini appear smaller than usual. Microscopically the change is seen to be due to a diminution in size of the liver cells, and hand-in-hand with this there occurs frequently an accumulation of pigment granules within the atrophied cells. The cells may entirely disappear over circumscribed areas, leaving only shrivelled blood-vessels and connective tissue; or, in some cases, there may be an increase of connective tissue in connection with the atrophy of the cells. When much pigment is formed in the cells, the lesion is often called *pigment atrophy*.

Essentially the same changes may occur in circumscribed portions of the liver, as the result of pressure from new connective tissue in cirrhosis, from tumors, hydatids, amyloid degeneration, gall stones, etc. In atrophy from pressure, the liver cells are apt to become very much flattened and squeezed together as they diminish in size.

DEGENERATIVE CHANGES.

Parenchymatous Degeneration (Cloudy Swelling).—In a variety of acute and infectious diseases—pneumonia, typhoid and typhus fevers, scarlatina, variola, diphtheria, erysipelas, yellow fever, septicæmia, and in certain cases of acute anæmia and phosphorus poisoning—the liver is somewhat swollen and, on section, of a dull yellowish-gray color, looking somewhat as if it had been boiled. It contains less blood than usual, and the outlines of the lobules are indistinct. Microscopical examination shows the lesion to consist of a swelling of the liver cells and an accumulation in them of moderately refractile, finer and coarser albuminous granules. Those granules may disappear and the cells return to their normal condition, or, as is frequently the case, they may pass into a condition of fatty degeneration. Very frequently fatty and parenchymatous degenerations are associated together.

Fatty Infiltration.—In the normal human liver, there is usual certain amount of fat in the liver cells, and this amount varies con erably under different conditions.

The gross appearance of pathological fatty livers varies a good d depending upon the amount and distribution of fat and its associa with other changes. If the lesion is uncomplicated and considera the organ is increased in size, the edges rounded, the consistence fi the color yellowish, and the cut surface greasy. The lobules are larged and their outlines usually indistinct, and the blood-con diminished. The liver is increased in weight. If the amount of i tration be moderate, the outlines of the lobules may be more disti than usual, and the centres appear unusually red. This is due to fact that the accumulation of fat usually commences in the periph of the lobules and progresses towards the centres, so that the cen appears darker by contrast with the fatty periphery. The lesion ma uniform throughout the organ or it may occur in patches. In the l

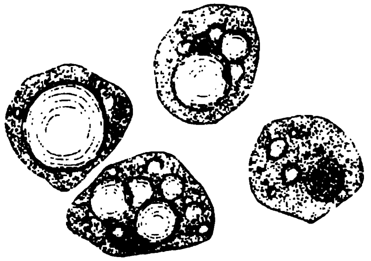

FIG. 162.—FATTY INFILTRATION OF LIVER CELLS.

ter case, the liver has a mottled appearance, irregular yellowish patch alternating with the brownish-red, unaffected portions.

Fatty infiltration is often associated with chronic congestion (*nutm liver*), with cirrhosis and amyloid degeneration; the picture may the present considerable complexity. Fatty livers may be stained brown greenish with bile pigment.

Microscopically the liver cells are seen to contain larger and small droplets of fat (Fig. 162), and frequently large drops of fat occupy near the entire volume of the cell, so that the protoplasm may be visible onl as a narrow, nucleated crescent at one side, or it may disappear alt gether. The microscopical appearances, of course, vary, depending upo the degree of infiltration and the association with other lesions.

Fatty infiltration of the liver may occur as a result of excessive i gestion of oleaginous food; in chronic alcohol, phosphorus, and arsen poisoning; in certain exhausting diseases accompanied by malnutritio as in pulmonary phthisis, chronic dysentery, etc., and under a variety conditions which we do not understand.

Fatty Degeneration.—In this condition, which in many cases cannot be morphologically distinguished from fatty infiltration, the fat is believed to be formed by a transformation of the protoplasm of the liver cells. The fat-droplets are, for the most part, very small and abundant, though this is not constant. Fatty degeneration of the liver cells frequently follows, and is associated with, cloudy swelling under the varying conditions in which this occurs, or it may appear in profound anæmia and in acute phosphorus and arsenic poisoning.

Amyloid Degeneration (Waxy Liver).—In the liver, amyloid degeneration may be general or local; so extensive as to give the organ very characteristic appearances, or so slight as to be unrecognizable without the aid of the microscope. It may be associated with other lesions. When the change is extensive and general, the liver is enlarged sometimes to more than twice its normal size; the edges are thickened and rounded; the surface smooth; the tissue tough, firm, inelastic, more or less translucent, and of a brownish-yellow color. The lobular structure may be more or less indistinct, or it may become very evident by an associated fatty degeneration of the peripheral or central cells of the lobules. The translucency and peculiar appearance of the tissue may be best seen by slicing off a thin section and holding it up to the light. When the lesion is less considerable, the liver may be of the usual size, and may feel harder than normal, and here and there a translucent mottling may be evident, or the degeneration may be apparent only on the addition of staining agents (see page 66). When, as is frequently the case, it is associated with cirrhosis, the liver may be small and nodular, and the appearance of the cut surface will vary greatly, depending upon the character of the cirrhotic change and the presence or absence of fat.

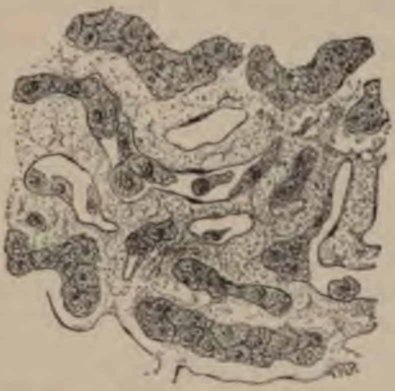

FIG. 163.—AMYLOID DEGENERATION OF LIVER CAPILLARIES.

This degeneration usually commences in the walls of the intralob blood-vessels, causing them to become thickened and translucent. liver cells are squeezed by the thickening of the vessels and may bec partially or completely atrophied (Fig. 163).

It is stated by some observers that the liver cells may also beco waxy, but we have been unable to find them unmistakably thus chang The liver cells not infrequently undergo fatty metamorphosis. Amyl degeneration may also involve the interlobular vessels, and in advan stages larger and smaller areas of liver tissue may be nearly or complet converted into the dense, refractile substance which in its arrangem but obscurely represents the grouping and structure of the affec lobules. Not infrequently atrophic or fatty liver cells are seen scatte singly or in clusters through the amyloid masses. In the affected regi the blood-content of the liver is considerably diminished, or it may nearly entirely absent.

Amyloid degeneration of the liver is usually associated with a simil lesion of other organs, such as spleen, kidneys, intestines, etc., althou it may occur in this organ alone. It usually occurs in cachectic con tions, as in chronic phthisis; in chronic suppurations, especially of t bones; in syphilis, and sometimes in malarial poisoning. It occasional occurs unassociated with any of these conditions.

PIGMENTATION OF THE LIVER.

As a result of severe malarial poisoning, a variable amount of brow black, or reddish pigment is often found in the blood. This is usual mostly taken up by the leucocytes and deposited in various parts of th body, chiefly in the liver, spleen, and marrow of the bones. In the live it is usually found inclosed in variously shaped cells, which lie especiall in the blood-vessels, but sometimes in the tissue between them (see Fig 164). The liver cells frequently contain bile pigment, but usually ar free from the melanotic pigment characteristic of this malarial condition As the result of this accumulation of pigment, the liver may have a dark reddish-brown, an olive-brown, or black color (sometimes called *bronz*

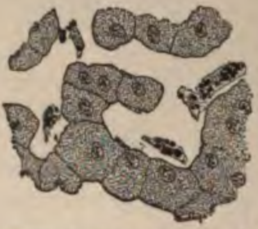

FIG. 164.—PIGMENTATION OF THE LIVER IN MALARIAL FEVER.
The pigment in this specimen was contained in cells lying within the liver capillaries.

liver). This condition may be associated with various other lesions of the liver, depending upon the nature and extent of which the organ will present a great variety of appearances. Thus there may be fatty or waxy degeneration, cirrhosis, chronic congestion, etc.

Pigment may be found in the connective tissue along the portal vessels similar in character to that which occurs in the lungs from the inhalation of coal dust. This inhaled pigment, according to the researches of Weigert, doubtless finds access to the blood (see page 60), and is deposited in the liver as it is in the spleen and hepatic lymph nodes.

Pigmentation of the liver cells, which is to a certain extent normal, may be greatly increased as a result of atrophy, localized hæmorrhage, and of obstructive jaundice.[1]

ACUTE YELLOW ATROPHY OF THE LIVER.

This disease is characterized anatomically by a rapid diminution in the size of the liver as the result of a granular and fatty degeneration and disintegration of the liver cells. The liver, sometimes within a few days, may be reduced to one-half its normal size. On opening the abdominal cavity, the organ may be found lying concealed by the diaphragm, close against the vertebral column. The amount of diminution and the general appearance of the affected organ depend to a considerable extent upon its previous condition—*i.e.*, whether or not it was the seat of other lesions—as well as upon the degree of degenerative change. In general, if the lesion is well marked, the liver is small, flabby—sometimes almost fluctuating—and the capsule wrinkled. On section, the cut surface may show but little trace of lobular structure, but presents an irregular mottling with gray, ochre-yellow, or red; sometimes one, sometimes another color preponderating.

Microscopical examination shows varying degrees of degeneration and destruction of the liver cells. Most evidently in those parts which have a grayish appearance, the outlines of the cells are preserved and the protoplasm is filled with larger and smaller granules. In the yellow portions, the outlines of the liver cells may be preserved, and they may contain varying quantities of larger and smaller fat-droplets and granules of yellow pigment. Or the cells may be completely disintegrated, and in their place irregular collections of fat-droplets, pigment granules, red and yellow crystals, and detritus; only the connective tissue and blood-vessels of the original liver tissue remaining. The red areas may show nearly complete absence of liver cells and cell detritus, and sometimes irregular rows of cells which are variously interpreted as being new-formed gall ducts or proliferated liver cells. In these areas it appears

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[1] The distribution and amount of the pigment may be well seen by staining thin sections with eosin and mounting in eosin-glycerin or balsam.

canal, kidneys, bladder, and lungs. There is usually marked jau
Rod-shaped bacteria and micrococci have been found in the live
their significance is doubtful; we have not been able to find them
cases which we have examined. The cause of the disease is unk
and it is doubtful whether it is a disease primarily of the liver, or
eral disease with local lesions. It is not unlikely that more tha
form of lesion is grouped under this heading.

INFLAMMATION OF THE LIVER.

Acute Hepatitis (Purulent Hepatitis; Abscess of the Liver).—
lent or suppurative inflammation of the liver may be the result of i
it may be secondary to inflammation of the gall ducts or the br
of the portal vein. It may occur as the result of the presence of t
parasites, or from propagation of an inflammatory process from wi
as in ulcer of the stomach with adhesions to the liver and second
volvement of the latter. It is probably directly due to the introd
into the organ, through the blood-vessels or otherwise, of ba
Purulent inflammation in the liver almost always results in absce

Large abscesses of the liver may be traumatic, but are, for th
part, due to unknown causes. They are not infrequently associate
dysentery, and may then be due to the conveyance through the v
septic material from the intestinal ulcers. They occur most freq
in tropical climates, but are not very uncommon in the temperate
They are usually single, but there may be several of them. T
sometimes so large as to occupy a large part of a lobe. They ar
frequent in the right lobe, but may occur in any part of the
They tend to enlarge, and as they do so they approach the surface

The early stages in the formation of large abscesses of the liver are but little known. It is probable, however, that in many cases they are the result of the confluence of smaller abscesses. Their contents, usually bad smelling, may be thick and yellow, like ordinary pus; but more commonly they are thin, reddish brown or greenish in color, from admixture with the pus of blood, gall pigment, and broken-down liver tissue. Microscopical examination shows the contents to consist of fluid with pus cells, more or less degenerated blood, degenerated liver cells, fragments of blood-vessels, and pigment granules and crystals. The walls of the abscess are usually ragged, shreds of necrotic liver tissue hanging from the sides. Microscopical examination of the liver tissue near the abscess shows infiltration with pus, flattening of the liver cells from pressure, cloudy swelling, and necrosis of those lying along the cavity.

After the discharge of the contents of the abscess, or without this if it be not very large, granulation tissue may form in the wall of the cavity, and a fibrous capsule be produced, inclosing the contents, which become thickened and often calcareous, and in this condition may remain for a long time. Or the connective-tissue walls may approach one another and join, forming a fibrous cicatrix at the seat of the abscess.

Abscesses of the liver accompanying inflammation of the portal vein and gall ducts are considered elsewhere in this section.

Small *multiple metastatic abscesses* are not infrequent in pyæmia, and are called *pyæmic abscesses*. In these abscesses we can readily study the various stages of formation. Suppurative processes in any part of the body—in the head, upper and lower extremities, etc.—may favor the production of the noxious materials, which in most cases are probably associated with bacteria, or are bacteria themselves. These, entering the circulation, may pass the heart and pulmonary capillaries, with or without inducing lesions in the lungs, and, lodging in the vessels of the liver, induce circumscribed necrosis of the liver tissue and suppurative inflammation. Under these conditions, we may find on a section of the liver larger and smaller yellowish or grayish spots, the larger of which may be soft and present the usual characters of abscesses. The smaller, which may not be larger than a pin's head, may present the usual consistence of liver tissue with the lobular structure still evident, others may be softer, more yellow, and surrounded by a zone of hyperæmic liver tissue. Microscopical examination of the earlier stages often shows the blood-vessels filled with micrococci, scattered and in masses. Around these the liver cells are found in various stages of necrosis; in many the nuclei do not stain, and the bodies are very granular; or the entire cell is broken down into a mass of detritus. About these necrotic islets of liver cells pus cells collect and often form a zone of dense infiltration. Thus, by the increase of pus cells and the necrosis of liver tissue, small abscesses are formed, whose contents are intermingled with greater or less quantities of bacteria, which seem to increase in num as the process goes on. By the confluence of small abscesses, larger c may be formed. Death usually ensues, however, before the abscesse tain a very large size.

Chronic Interstitial Hepatitis (Cirrhosis).—The primary result chronic interstitial hepatitis is the formation of new connective ti in the liver. The character, amount, and distribution of the new ti vary greatly in different cases. Secondarily there are usually mar

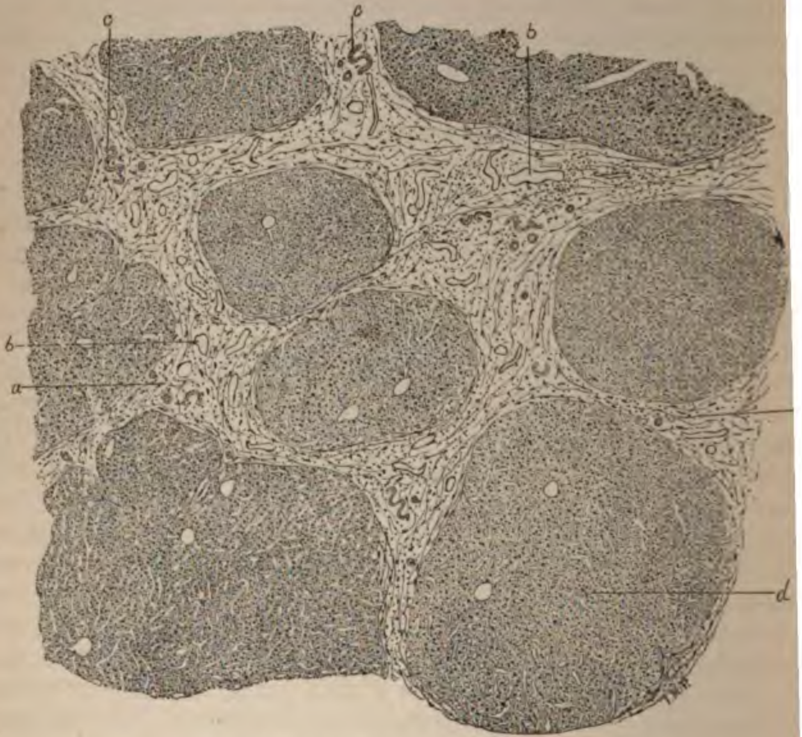

FIG. 165.—CHRONIC INTERSTITIAL HEPATITIS.

a, new-formed connective tissue; *b*, dilated blood-vessels of the new tissue; *c*, gall du *d*, parenchyma of liver.

changes in the liver cells and in the blood-vessels and gall ducts. T new tissue is most commonly formed and most abundant in the pe phery of the lobules along the so called capsule of Glisson, but it m extend into the lobules between the liver cells. It may surround sing lobules, or more frequently larger and smaller groups of lobules (Fi 165). It may occur in broad or narrow, irregular streaks or bands. It frequently more abundant in one part of the liver than in another. Th new-formed tissue tends to contract, and thus compromise by pressure the inclosed islets of liver tissue, causing them to project, in larger and smaller nodules, from the surface of the organ. The liver cells may be flattened or atrophied from pressure; or, from interference with the portal circulation, they may atrophy or become fatty; or they may become colored with bile pigment. The varied appearances which cirrhotic livers present to the naked eye depend largely upon the amount and distribution of the new connective tissue, and upon the secondary changes in the liver cells.

In some cases the liver is enlarged, sometimes so much so as to weigh nine or ten pounds, the surface smooth or slightly roughened; in other cases it may be finely or coarsely nodular on the surface. It may be smaller than normal, sometimes very small indeed, so as to weigh only one or two pounds. The surface may then be very rough and uneven from the projection of larger and smaller nodules of liver tissue, or it may be quite smooth; or the organ may be greatly distorted by the contraction of large bands or masses of new connective tissue. In section through cirrhotic livers, the new tissue may not be visible to the naked eye, or it may appear as grayish, irregular streaks, or bands, or patches, often sharply outlined against the dark-red, or brown, or yellow, or greenish-yellow parenchyma.

On microscopical examination, the new connective tissue is found in some cases loose in texture, and containing many variously shaped cells; or it may be dense and contain comparatively few cells; it is usually quite vascular. Not infrequently, when occurring largely between the lobules, it will be found to have incroached more or less upon their peripheral portions. Very frequently there are found in the new connective tissue cylindrical ducts lined with cuboidal cells, and resembling gall ducts (Fig. 166, *c*); or irregular rows of more or less cuboidal or polyhedral cells, which look somewhat like the lining cells of the medium-sized gall ducts, or like altered liver cells. The branches of the hepatic and portal veins, particularly the latter, often become obliterated by pressure from the new connective tissue, or from chronic thickening of their walls, so as to seriously interfere with the function and nutrition of the liver cells. The bile ducts also may become obliterated, or there may be catarrhal inflammation, especially of the larger trunks. The branches of the hepatic artery are much less liable to alterations than the other vessels. The capsule of the liver is usually thickened either uniformly or in irregular patches; or its surface may be roughened by larger and smaller papillary projections. The liver is frequently bound to the diaphragm or other adjacent organs by connective-tissue adhesions. Amyloid and fatty degeneration may be associated with cirrhosis. Cirrhotic livers frequently show an unusual number of leucocytes in the blood-vessels.

The obstruction to the portal circulation induced by cirrhosis usua gives rise to a number of secondary lesions, since collateral circulat is rarely established in sufficient degree to afford much relief. T hæmorrhoidal and vesical veins may be greatly enlarged, and also ve of communication between Glisson's capsule and the diaphragma veins.

In rare cases, a very peculiar dilatation of the cutaneous veins ab the umbilicus is observed. The enlarged veins form a circular netw around the umbilicus, or a pyramidal tumor alongside of it, or all t veins of the abdominal wall, from the epigastrium to the inguinal regi are dilated. This condition is said to be produced by the congeni non-closure and subsequent dilatation of the umbilical vein and its ana tomoses with the internal mammary, epigastric, and cutaneous vein According to Sappey, it is not the umbilical vein which is dilated, but vein which accompanies the ligamentum teres.

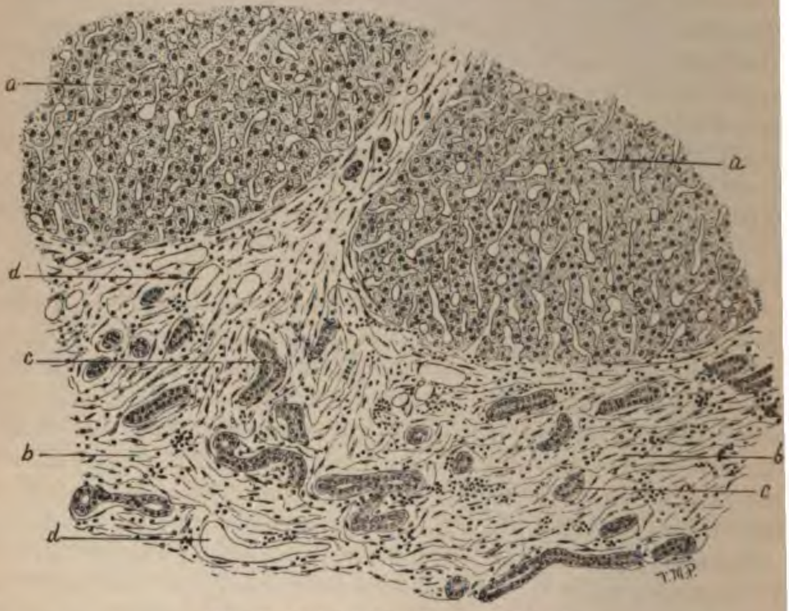

FIG. 166.—CHRONIC INTERSTITIAL HEPATITIS.

Showing a portion of the section shown in Fig. 165, but more highly magnified. *a*, portions liver lobules; *b*, new-formed connective tissue; *c*, gall ducts, apparently new formed; *d*, blo vessels in the new tissue.

There is very frequently also a dilatation of the veins of the abdom nal wall, which has a different cause. It is produced by the pressure the fluid of ascites on the vena cava, and is found with ascites from an cause and with abdominal tumors.

Ascites is the most common secondary lesion of cirrhosis. It usuall begins at an early stage of the disease, and is apt to increase constantly. It usually precedes œdema of the feet, but both may appear at the same time. This fluid is of a clear yellow or brown, green or red; it is sometimes mixed with shreds of fibrin, and more rarely with blood. The peritoneum remains normal, or becomes opaque and thick, or there may be adhesions between the viscera.

The spleen is very frequently enlarged, and the enlargement may be very considerable. When it is not increased in size, this seems usually due to previous atrophy of the organ, or to fibrous thickening of its capsule, or to hæmorrhages from the stomach and bowels, occurring just before death.

The stomach and intestines are often secondarily affected by the obstruction to the portal circulation. Profuse hæmorrhage from the stomach and intestines may occur, and sometimes cause sudden death. The mucous membrane is then found pale, or congested, or with hæmorrhagic erosions. Sometimes the blood is infiltrated in the coats of the stomach and intestines. The mucous membrane of the stomach, and of the entire length of the intestines, is frequently the seat of chronic catarrhal inflammation, and is sometimes uniformly and intensely congested and coated with mucus. In other cases both the mucous and muscular coats are pale, but very markedly thickened.

Cirrhosis of the liver is not infrequently accompanied by chronic diffuse nephritis.

The causes of cirrhosis are imperfectly understood. It is a disease of adult life, but exceptionally occurs in children. In adults, it seems in many cases to be directly dependent upon the continued ingestion of large quantities of strong alcoholic liquors. It very rarely occurs as a result of beer drinking. There are many cases of cirrhosis for which no cause can be discovered.

Syphilitic Hepatitis.—Chronic interstitial inflammation of the liver very frequently results from syphilitic infection, either congenitally or in the later stages of the acquired form. It may occur in a diffuse manner, new connective tissue being formed either between the lobules, or within them between the rows of liver cells. The new tissue may be rich in cells, or dense and firm. This form is frequently seen in children, and cannot be distinguished, either macroscopically or microscopically, from similar forms of interstitial hepatitis from other causes.

In other cases, particularly in children, there may be numerous small gummata (so-called *miliary gummata*) scattered through the liver, together with more or less new connective tissue (Fig. 167). In adults, gummata are usually larger, varying in size from that of a pea to a hen's egg, and may be surrounded by larger and smaller irregular zones of ordinary connective tissue (Fig. 168). In still other cases in adults, we find larger and smaller dense, irregular bands or masses of connective tissue running through the liver, drawing in the capsule, and of causing great deformity of the organ. These bands and masses of n tissue may or may not inclose gummata, either large or small. Th deforming cicatrices, either with or without gummata, are very char teristic of syphilitic inflammation of the liver.

This, like the simple interstitial inflammation of the liver, may associated with fatty and waxy degeneration, and with atrophy of parenchyma from pressure.

Tubercular Hepatitis.—This lesion, which is usually secondary tubercular inflammation in some other part of the body, or a part c acute general miliary tuberculosis, is most frequently characterized b the formation of larger and smaller miliary tubercles, which may b either within or between the liver lobules, or in the walls of the bil ducts. Many of the tubercles are too small to be seen with the nake eye; others may be just visible as grayish points; still others may be fron one to three mm. in diameter, with distinct yellowish-white centres Microscopical examination shows considerable variation in the structur of the tubercles in different cases, as well as in the same liver. Some o them, usually the smaller ones, consist simply of more or less circum

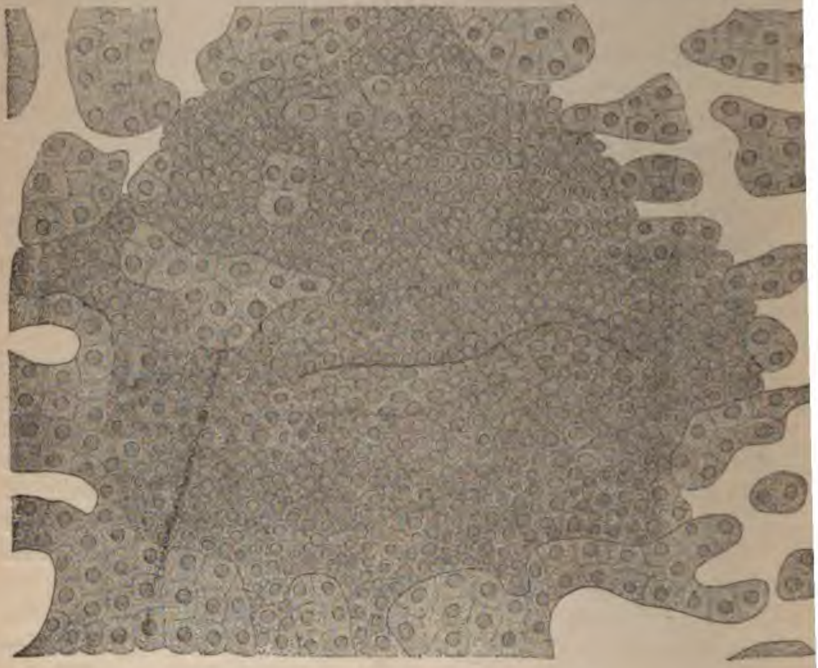

FIG. 167.—SYPHILITIC HEPATITIS.

A so-called miliary gumma from the liver of a child with congenital syphilis.

scribed collections of small spheroidal cells, which are not morphologically distinguishable, so far as the form and arrangement of the cells are concerned, from simple inflammatory foci, or from the diffuse masses of lymphatic tissue which occur normally in the liver.

In other forms we find a well-marked reticulum with larger and smaller spheroidal and polyhedral cells, with or without giant cells. In still other forms there is more or less extensive cheesy degeneration. The larger forms are conglomerate, being composed of several tubercle granula joined together to form a single nodular mass. The liver cells at the seat of the tubercle are destroyed, and the interstitial tissue and blood-vessels either destroyed or merged into the tubercle tissue. In the periphery of the tubercles the liver cells may be in a condition of coagulation necrosis, and the tissue round about may be infiltrated with small spheroidal cells. There is in some cases a new formation of gall ducts or of structures which resemble these, and which in transverse sections look considerably like giant cells. Tubercle bacilli, frequently in small numbers, but often in great abundance, may be found within the tubercles.

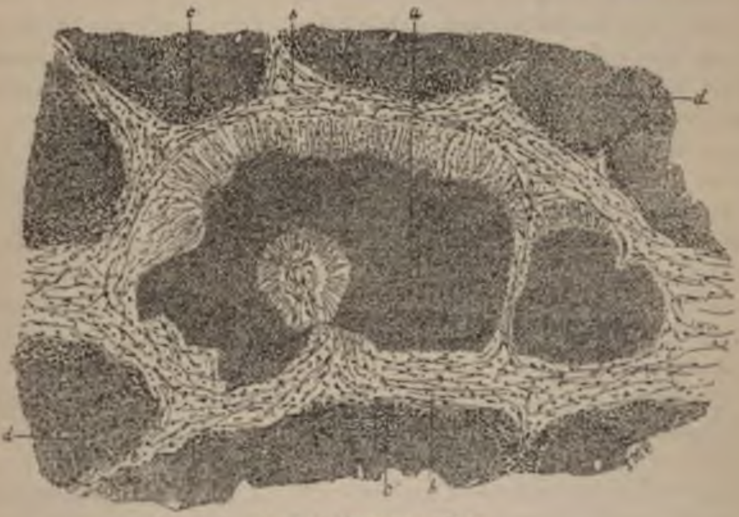

FIG. 168.—GUMMA OF LIVER.

a, cheesy centre; *b*, fibrous periphery; *c*, small-celled peripheral infiltration; *d*, portions of liver lobules.

Tuberculosis of the liver may be associated with cirrhosis, waxy and fatty degeneration.

Much more rarely than the above form there are found in the liver more or less numerous scattered tubercular masses from the size of a pea to that of a walnut or larger, with cheesy centres and usually a new growth of connective tissue in the periphery. These so-called *solitary* *tubercles* of the liver may be softened at the centres. Tubercular infl mation of the gall ducts may give rise to numerous scattered, che nodules, as large as a pea or larger, which may be softened at the cen and stained yellow with bile. This lesion is rare and seems to be m frequent in children than in adults

Perihepatitis.—Acute inflammation of the serous covering of liver, with the formation of fibrin, may occur as a part of acute genera localized peritonitis, and over the surface of abscesses, tumors, hydati etc., of the organ, when these lie near or approach the surface; or it n be secondary to acute pleurisy.

Chronic perihepatitis, resulting in the thickening of and formation new connective tissue in and beneath the capsule of the liver, may secondary to an acute inflammation of the capsule, or it may be chro from the beginning and associated with chronic pleurisy, chronic pe tonitis, and cirrhosis. In this way, more or less extensive adhesions the liver to adjacent structures may be formed; or, by contraction of t new-formed connective tissue, considerable deformity of the liver m be produced. The capsule is sometimes uniformly thickened, sometim the new tissue occurs in more or less sharply circumscribed patch The surface is sometimes roughened from little, irregular projecti masses of connective tissue. Microscopically the new-formed tissue usually dense and firm, but it may be loose in texture and contain ma cells. Not infrequently bands or masses of connective tissue run inwa from the thickened capsule between the superficial lobules, causing loca ized atrophy of the parenchyma.

Hyperplasia of Lymphatic Tissue in the Liver.—In some forms leukæmia and pseudo-leukæmia, the liver is not infrequently enlarg and soft and besprinkled with small white spots, or streaked with narro whitish, irregular bands, or of a diffuse grayish color. Microscopical e amination shows this change to be due to an accumulation of cells r sembling leucocytes, either along the portal vein, or diffusely throug the liver tissue, or in small circumscribed masses. The amount of accu mulation of these small cells varies much, but is sometimes so great as t seriously compromise the liver cells. The origin of these new cells is no yet definitely known. They may be, and doubtless in part are, brough to the organ through the portal vein, but they may, in part at least, b formed in the liver itself.

In typhoid fever, small-pox, scarlatina, diphtheria, and measles, small circumscribed masses of cells resembling leucocytes are sometimes found in the liver, lying in the meshes of a delicate reticular tissue. These are sometimes called *miliary lymphomata;* but it should be remembered (see p. 301) that small masses of lymphatic tissue normally occur in the liver, and that as, under the above conditions, an hyperplasia of the lymph nodes and spleen is wont to occur, these so-called lymphomata are very probably normal structures, which have become more prominent under the conditions of disease owing to an acute inflammatory condition induced by absorbed ptomaïnes.

TUMORS OF THE LIVER.

Tumors of the liver may be primary or secondary; the latter are most common.

Cavernous Angiomata.—These tumors, usually small, from five to fifteen mm. in diameter, are most common in elderly persons and are of no practical significance. They may be situated at the surface or imbedded in the organ, and are of a dark-red color; sometimes sharply circumscribed by a connective-tissue capsule, sometimes merging imperceptibly into the adjacent liver tissue. Microscopically they consist of a congeries of irregular cavities (Fig. 66, page 145), filled with blood and frequently communicating freely with one another. The walls of the cavities consist of connective tissue, often containing small blood-vessels, and are sometimes thick, sometimes thin. They are believed to be formed by dilatation of the liver capillaries, with subsequent thickening of their walls and atrophy of the adjacent liver cells.

Small *fibromata* and *lipomata* have been described, as also *fibro-neuromata* of the sympathetic.

Adenomata of the liver are of not infrequent occurrence. They are sometimes small and circumscribed, sometimes very large and multiple. They present two tolerably distinct types of structure. In one form the tissue presents essentially the same structure as normal liver tissue, except that the arrangement of the cells is less uniform and the cells are apt to be larger. They look like little islets of liver tissue, sometimes incapsulated and sometimes not, lying in the liver parenchyma. In the other form, the cells are less like liver cells, are frequently cylindrical, and are arranged in the form of irregular masses of tubular structures with more or less well defined lumina. These tumors are sometimes large and multiple, and in one case described by Greenfield there were metastatic tumors in the lungs. These tubular adenomata are in some cases so closely similar to some of the carcinomata as to be scarcely distinguishable from them, and seem, indeed, to merge into them.

Carcinomata are the most common and important of the liver tumors, and may be primary or secondary. Primary carcinomata of the liver are probably developed from the epithelium of the gall ducts, and in some cases are arranged along the larger trunks. Their cells are usually polyhedral, sometimes cylindrical, and may be arranged irregularly in alveoli or form more or less well-defined tubular structures.

Secondary carcinomata of the liver, which are by far the most common, are most frequently due to the dissemination in the organ of tumor cells from carcinomata of the stomach, intestines, pancreas, or gall bladder. But they may be the result of metastases from the mamma, phagus, uterus, and various other parts of the body. In secondary cinomata, the cells resemble more or less closely the type of those forn the primary tumor.

The form in which the carcinomatous nodules in the liver pre themselves is subject to considerable variation. Sometimes they single, but more often multiple; they may be very large, or so smal to be scarcely visible to the naked eye; very frequently numerous sr nodules are grouped in the periphery of a larger cancerous mass. T are sometimes deeply imbedded in the liver, sometimes they pro from the surface. The liver is frequently enlarged, sometimes er mously so. The nodules are usually whitish, or yellowish, or pink color, but they are often the seat of hæmorrhages, and may become s ened at the centre, forming cysts filled with degenerated tumor tis which is often mixed with blood. The nodules are sometimes ha sometimes soft and almost diffluent. Fatty degeneration is freque and may be evident to the naked eye in the form of yellowish streaks patches on the cut surfaces. Owing to the degeneration and par absorption of the central portions of the tumors, the nodules on surface frequently present a shallow depression at the centre T tumors may be sharply outlined against the adjacent liver tissue, or m merge imperceptibly into it. They may be so large or numerous as occupy the greater part of the enlarged organ. The liver tissue in th vicinity shows flattening and atrophy of the liver cells from pressu and there may be infiltration with small spheroidal cells. The tum may press upon the portal vein or its branches, or upon the gall duc and thus seriously interfere with the functions of the organ. Sometim however, the tumors are very large and abundant without causing a apparent detriment to the liver functions. They are not infrequent stained with bile. *Melanotic carcinomata* sometimes occur in the live most frequently as secondary tumors.

In some cases, instead of forming separate, distinct nodules, the ca cerous growth develops in the form of a diffuse infiltration of the orga so that the often greatly enlarged liver is irregularly mottled with whi and reddish-brown masses, and may then somewhat resemble some forn of chronic interstitial hepatitis.

Sarcomata.—Spindle-celled, melanotic, and telangiectatic sarcoma may occur in the liver as secondary tumors. Secondary *myxomata* a *chondromata* have also been described, but they are very rare.

Cavernous lymphangiomata have been described in a few case *Cysts*, usually of small size, may occur by dilatation of the bile duct They may be multiple and contain serum, mucus, and degenerat epithelium. Single cysts, apparently unconnected with the gall duct are occasionally found in the connective tissue of the liver. They may be lined with ciliated epithelium.

The liver is sometimes the seat of larger and smaller *multiple cysts*, varying from microscopical size up to that of a pea, and sometimes larger. They do not appear to communicate with the gall ducts. They are sometimes associated with multiple cysts of the kidney. Their origin and nature are not understood.[1]

Occasionally the liver is found at the autopsy, even if this be made but a few hours after death, more or less completely riddled with small, irregular-shaped cavities, from the size of a pin's head to that of a pea. These holes appear to be due to the accumulation of putrefactive gases in the liver, and close about them, as well as in the blood-vessels, large bacilli and other forms of bacteria may be found.[2]

PARASITES.

Echinococcus.—This parasite is the most common and important of those which occur in the human liver. It forms the so-called *hydatids* of the liver. These represent one of the developmental stages of the small tape-worm of the dog, *Tænia echinococcus* (see page 73). The cysts in the liver may be very small and multiple, but they may be as large as a man's head or larger. The liver may be greatly increased in size, and the tissue about the cysts atrophied. The liver itself furnishes a connective-tissue capsule, within which is the translucent, lamellated membrane furnished by the parasite. On the inside of this we may find a layer of cells, granular matter, and a vascular and muscular system belonging to the parasite. Projecting from this inner capsule are the brood capsules and heads or scolices of the immature tape-worm. The scolices may become detached from the wall and lie free in the cavity, which is filled with a transparent or turbid fluid. Not infrequently the cysts are sterile, and are then simply filled with clear or turbid fluid; or the embryos may have died and disintegrated, and their detritus, including the booklets, may be intermingled with the fluid contents of the cysts. The contents of the cysts may be mixed with fat, cholestearin crystals, pus, bile, or blood; or form a grumous mass, in which we may or may not be able to find the hooklets of the scolices or fragments of the lamellated wall. The connective tissue of the walls of the cysts may be greatly thickened or they may be calcified.

In other countries the lesion is much more common and frequently more formidable than in the United States. The cysts reach an enormous size, the veins of the liver may be compressed and filled with thrombi, the bile ducts compressed and ulcerated. So much of the liver tis may be replaced by the hydatids that the patient may die from t cause alone. Very frequently there is local peritonitis, and adhesi are formed between the liver and the surrounding parts. In some ca the cysts rupture, and their contents are emptied into the periton cavity, the stomach, the intestines, the pleural cavity, or the lung tiss Sometimes the cysts perforate the bile ducts, the vena cava, or some the branches of the portal or hepatic veins. Sometimes the abdomi wall is perforated and a fistula formed between the cavity in the li and the surface.

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[1] Consult *Pye-Smith*, "Cystic Disease of Liver and both Kidneys." Trans. London Path. Soc., vol. xxxii., p. 112, 1881.

[2] *Freeman*, Trans. New York Path. Society, 1889.

In cases in which we do not find the scolices entire, a careful exa nation of the inner cyst wall or of its contents will frequently establi

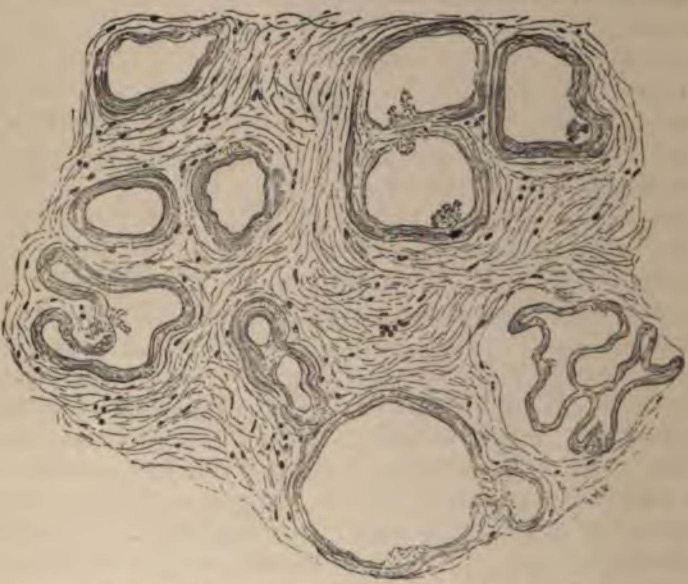

FIG. 169.—ECHINOCOCCUS MULTILOCULARIS OF THE LIVER.

the diagnosis by revealing single hooklets (see Fig. 13, page 75) or fragments of the characteristically lamellated wall (see Fig. 11, page 74).

Echinococcus multilocularis, which is apparently an abortive form of the above species (see page 75), is very rare indeed in the United States. The writer (T. M. P.) has examined a specimen sent to him by Dr. Edward J. Ill, of Newark, N. J., and which is now in the museum of the College of Physicians and Surgeons, New York. The patient was a male, age thirty-one, German, single, farmer. He had been in the United States five years. For a year previous to his death he had been out of health, and jaundiced, and somewhat emaciated. A large, indistinctly fluctuating tumor was evident in the right lumbar and umbilical regions, and apparently connected with the liver. Aspiration of the tumor gave a milky fluid believed to be pus. An opening was made into the tumor by one of the surgeons attending the case, and death occurred, after ten hours, from hæmorrhage.

The liver was found adherent to the abdominal walls, and about one-fourth of the right lobe of the liver was occupied by an irregular cavity with very rough, ragged walls. These walls were in some places from one to two inches in thickness, and appeared to the naked eye to consist of dense connective tissue in irregular bands and fascicles, which inclosed very irregular, mostly small cavities. Microscopical examination showed that the cavities were lined with the delicate, lamellated cuticula characteristic of the echinococcus cysts. No hooklets were found. Fig. 169 is a drawing from this specimen.

Distoma hepaticum, *D. sinense*, *D. lanceolatum*, may occur in the gall ducts and gall bladder. *D. sinense* occurs especially in the East, and has been found in great numbers in the bodies of Chinamen. *D. hæmatobium* is very common in Egypt and Abyssinia, occurring in the blood-vessels of the liver.

Pentastoma denticulatum is the undeveloped form of Pentastoma tænioides, a parasite which inhabits the nasal cavity of dogs and some other animals. In the liver of man, it usually occurs in the form of small rounded, calcified cysts. The cysts may contain fat, calcareous matter, and the remains of the dead parasite, among which the hooklets may be found.

Ascaris lumbricoides sometimes finds its way from the intestines into the bile ducts. It may cause no disturbance here, but in some cases the worms have been present in large numbers, and caused occlusion, dilatation, and ulceration of the biliary passages, and have led to the formation of abscess of the liver.

Psorospermiæ, the very common parasite in the rabbit's liver, has been found a few times in the liver of man.

THE BILIARY PASSAGES.

Catarrhal Inflammation most frequently attacks the lower portion of the common duct and the gall bladder. In the acute form, it usually leaves but few changes appreciable after death. An abnormal coating of mucus, and sometimes congestion of the blood-vessels, are almost the only post-mortem lesions. Owing to the swelling of the mucous membrane and the accumulation of mucus in the lumen, the ducts may be temporarily occluded; but this occlusion may not be evident after death. If, however, the inflammation becomes chronic, the walls of the bile ducts may become thickened and their lumina more or less permanently obstructed. In consequence of this, dilatation or ulceration of the bile ducts may ensue. Temporary obstruction of the bile ducts may produce marked pigmentation of the liver, owing to the accumulation of pi granules in the liver cells, particularly in the vicinity of the cap Glisson, and jaundice of the entire body.

The gall bladder may be inflamed by itself or in connection w flammation of the biliary passages. If the disease is chronic, tl of the bladder may be thickened; polypoid growths may occur mucosa; the duct may be occluded; dilatation, ulceration, the for of gall stones, calcification, and atrophy may ensue.

Inflammation of the stomach and duodenum, hyperæmia and i mation of the liver, concretions, and parasites, are the usual ca catarrhal inflammation of the biliary passages, but it may occur w these.

Suppurative and Croupous Inflammation may attack the bilia sages and produce infiltration of pus in their walls and purulent f their cavities; or flakes and tubular casts of fibrin on their walls; filtration of their walls with fibrin, and consequent ulceration. lesions occur most frequently in connection with obstruction of t ducts, and in typhoid and typhus fever, pyæmia, cholera, or the be due to the extension of inflammatory processes from without. also occur under unknown conditions. Suppurative inflammatic produce perforations of the ducts or bladder, with escape of bile an tonitis; or fistulous openings between the gall bladder and the duod colon, and stomach, or through the abdominal wall. Or the infl tion may extend to the liver tissue and produce abscesses. Und latter conditions, we may find a series of small abscesses ranged the walls of the suppurating gall ducts. In more advanced stag abscesses may become large and communicate with one another, a considerable portion of the liver may be occupied by a series o municating cavities with ragged walls, containing pus and detr liver tissue more or less tinged with bile.

These abscesses are apt to contain various forms of bacter whether they always or frequently stand in a causative relation lesion or not has not been definitely established.

Such abscesses may become more or less completely inclosed l nective-tissue walls. The portal vein may also become inflame perforations may be formed between it and the bile ducts.

Constrictions of the biliary passages may also be produced by tl causes.

Constriction and Occlusion may be produced by inflammation ducts themselves, by new growths in their walls, by calculi or p in their lumina, by changes in the hepatic tissue in chronic and hepatitis, by aneurisms, or by pressure on the duct from without tumors in the head of the pancreas, etc.

The obliteration of the smaller bile ducts produces no marked l

When the ductus communis, or the hepatic duct, is obstructed, the ducts throughout the liver are frequently dilated and the liver tissue bile-stained. The liver may undergo atrophy and the whole body be intensely jaundiced. When the cystic duct is obstructed, the gall bladder is dilated.

Dilatation of the bile ducts is usually produced by strictures in the ways just mentioned, or by calculi. When calculi have produced the dilatation, this condition may sometimes continue after they have found their way into the intestines. Sometimes, however, we meet with very marked dilatation of the bile ducts without being able to make out any present or past obstruction. The dilatation may affect only the common and hepatic ducts, or it may extend to the smaller ducts in the liver, which are then dilated uniformly or sacculated. They may contain bile, mucus, or calculi. The liver is at first enlarged, but may afterward atrophy. The gall bladder may be dilated in consequence of obstruction of the common or the cystic duct. In the latter case, it may reach an immense size and form a large tumor in the abdominal cavity. The dilatation is generally uniform, the bladder retaining its normal shape; sometimes, however, there are diverticula, which are usually produced by calculi. If the obstruction to the hepatic duct is incomplete or movable, the gall bladder may contain bile, and often calculi. If the obstruction is complete. the contained fluid may gradually lose its biliary character, and become a serous or mucous fluid of a light yellow color—*hydrops cystidis fellæ*. The walls of the bladder may be of normal thickness, or thinned, or thickened, or calcified. If the obstruction is due to a calculus, this may pass into the intestine and the gall bladder be suddenly emptied. Usually the bladder fills again, owing to its loss of contractile power.

Biliary Calculi.—These bodies are of common occurrence. They are found usually in the gall bladder, sometimes in the hepatic, cystic, and common ducts; less frequently in the small ducts of the liver. In the gall bladder from 1 to 7,800 calculi have been counted. They vary in size from that of a pin's head to that of a hen's egg, or they may be larger. Single gall stones are usually spheroidal or ovoidal; when multiple, they are usually flattened at the sides or faceted.

They may be composed :

1. Principally of *cholestearin*, and may be of pure white color, or tinged with various shades of yellow or brown by bile pigment. The fractured surface shows a radiating crystalline structure.

2. Of *cholestearin*, *bile pigment*, and salts of *calcium* and *magnesium*. These are usually dark-colored, brown, reddish-black, or green, and may be spheroidal or faceted, smooth or rough on the surface; the fractured surface is usually radiating crystalline. This is the most common form.

may be homogeneous, or lamellated, or crystalline.

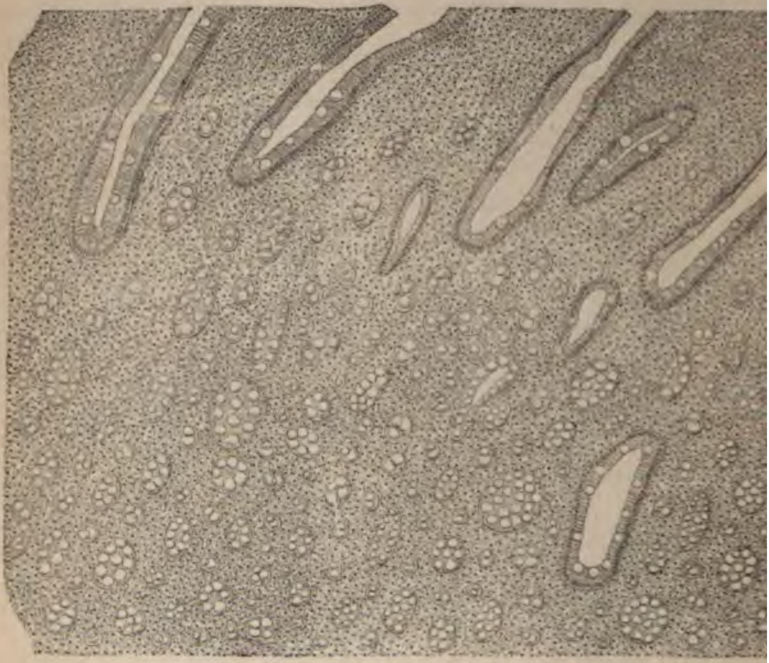

FIG. 170.—PRIMARY CARCINOMA OF THE COMMON BILE DUCT, × 300 and reduced.

Biliary calculi in the gall bladder may produce no symptoms an

TUMORS OF THE GALL BLADDER AND LARGER GALL DUCTS.

Small *fibromata* have been described in the gall bladder and in the common duct, but they are very rare. The most common tumors are *carcinomata*. These may be primary or secondary, and present the usual structural variations. The cells may be cylindrical, polyhedral, or they may present the characteristics of colloid cancer. Primary carcinomata of the gall bladder and larger ducts (Fig. 170) are not uncommon. Not infrequently the pancreatic and common ducts are both involved, and it is difficult to say whether the tumor is primary in the head of the pancreas or in the gall duct. The bladder and ducts may also be secondarily involved in carcinomata of the stomach, liver, and duodenum.

THE SPLEEN.

In studying the alterations produced in the spleen in disease, i important to bear in mind the peculiar relations in which this org stands to the blood-vessels and to the circulation. After passing throu the various branches of the splenic artery and the limited systems capillaries which are associated with it, the blood is not received at o into venous trunks, as in other parts of the body, but is poured direc into the pulp tissue. In this it circulates, under conditions which r der it liable to stagnation and undue accumulation, before it is tak again into well-defined vessels through the open walls of the cavern veins. Moreover, these conditions, naturally unfavorable to undisturb and vigorous circulation, are reinforced by the association of the sple with the sluggish and often interrupted portal circulation. Beari these considerations in mind, it will be in a measure plain why, as is fact the case, the spleen should be more liable to alterations in size th any other organ in the body, and why, serving as it does as a sort of blo filter, it should be especially susceptible to the influence of deleteric materials of various kinds which in one way or another gain access the blood. In this respect the relations of the spleen to the blood, a of the lymph nodes to the lymph, present suggestive analogies.

WOUNDS, RUPTURE, AND HÆMORRHAGE.

Wounds of the spleen are usually accompanied by extensive hæmo rhage, and are commonly fatal. Death usually occurs as the result this hæmorrhage, but it may be due to secondary inflammatory chang Healing and recovery may, however, occur.

Rupture of the spleen may be traumatic or spontaneous. In the fo mer case, it may be due to direct violence in the region of the organ to injury to the thorax, falls, etc. In certain diseased conditions, t spleen is more liable to rupture than when it is normal. The ruptu usually involves not only the capsule, but a more or less considerab portion of the parenchyma, and of course leads to hæmorrhage. Spo taneous rupture is rare, but may occur as the result of excessive enlarg ment of the organ, as in typhoid fever, malaria, etc.—see below—or as the result of abscess.

Hæmorrhage.—Aside from the extensive hæmorrhages from injury and rupture, the spleen may be the seat of small circumscribed hæmorrhages in various infectious diseases, although, owing to the peculiar distribution of the blood, it is often very difficult to distinguish between a moderate interstitial hæmorrhage and hyperæmia.

DISTURBANCES OF THE CIRCULATION.

Anæmia.—This may be associated with general anæmia, but it is not always present in this condition. When marked and unassociated with other lesions, the spleen is apt to be diminished in size, the capsule more or less wrinkled, the cut surface dry and lighter in color than normal, the trabeculæ unduly prominent.

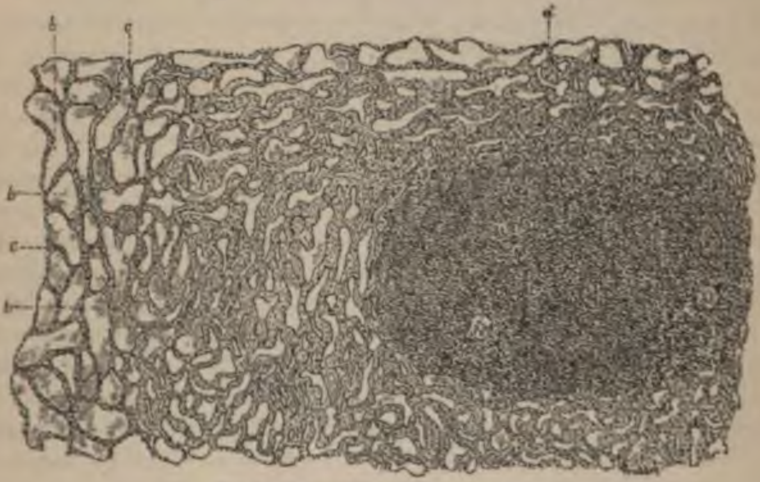

FIG. 171.—CONGESTION OF THE SPLEEN.

b, dilated cavernous veins; *c*, trabeculæ of pulp tissue compressed between dilated cavernous veins; *d*, glomerulus.

In this, as in other alterations simply of the blood-content of the spleen, neither the gross nor microscopical appearances are constant, because of the redistribution of blood which is apt to occur in the viscera after death.

Hyperæmia.—This may be passive, occurring when some obstruction to the portal circulation exists, most frequently in cirrhosis of the liver, but also with certain valvular lesions of the heart, emphysema, etc. The spleen is enlarged, but usually only to a moderate degree. The capsule is apt to be tense, and on section the pulp is dark red and may be soft or firm. The cavernous veins are dilated (see Fig. 171). Usually, when the lesion has existed for some time, there is a thickening of the trabeculæ and reticular framework of the spleen, so that they are pron nent on section. In other words, there is a chronic interstitial spleni following the chronic congestion.

Active Congestion of the spleen, which in most cases is scarcely to differentiated from some forms of acute inflammation, and probably many cases is associated with it, very frequently occurs in a great varie of acute and infectious diseases, such as typhoid fever, pneumoni diphtheria, pyæmia, the exanthemata, etc. The spleen is enlarged, t capsule tense; on section, the pulp is soft, dark red in color, often swe ing out from the cut surface and concealing the glomeruli and trabec læ. Under these conditions, we may find the cavernous veins distend with blood, and the interstices of the pulp infiltrated with a variabl sometimes large quantity of red and white blood-cells. Or we may fin in addition to this, an increase in cells, which characterizes acute i flammation or hyperplasia of the spleen (see below).

Infarctions of the Spleen.—Embolic infarctions of the spleen are frequent occurrence. They may be single or multiple, small or ve large, sometimes occupying half of the organ. They are in gener approximately wedge-shaped, corresponding to the area of tissue su plied by the occluded artery. They may be hæmorrhagic, *i.e.*, red, or th may be white (see page 57). Infarctions, originally red, may becon white after a time from changes in the blood-pigment. They m usually be seen as dark-red, reddish-white, or white, hard, sometim slightly projecting areas on the surface of the organ. Not infrequent the centre of the infarction is light in color, while the peripheral zone dark red. A layer of fresh fibrin is sometimes seen over the surface the infarction. The general as well as the microscopical appearanc which they present depend largely upon the age of the infarction. I the earlier stages, the hæmorrhagic infarctions present little more und the microscope than a compact mass of red blood-cells, among whic may be seen the compressed necrotic parenchyma. The white infarctic may show at first in a general way the usual splenic structure, but tl entire tissue is in a condition of coagulation necrosis. The tissue ma disintegrate and soften, and be more or less completely absorbed, wit or without fatty degeneration. A zone of inflammatory tissue ma appear around the infarction and upon the capsule, and this tissue, b coming denser, assumes the characters of cicatricial tissue, and contrac around the unabsorbed remnant of the infarction, so that finally nothin may be left but a dense mass of fibrous tissue, which frequently draws i the surface, causing more or less distortion of the organ. This cicatri may be pigmented or white.

If the embolus be of an infectious, irritating nature, in addition t its mechanical effects there may be suppuration, gangrene, and th formation of abscess. There may be perforation of the capsule and fatal peritonitis.

INFLAMMATION.

Acute Hyperplastic Splenitis (Acute Splenic Tumor).—The conditions under which acute inflammation of the spleen occurs have already been mentioned under active hyperæmia, with which it is usually associated. It is a frequent though not a constant accompaniment of the acute infectious diseases, and seems in all cases to be a secondary lesion. The spleen is enlarged, sometimes to two or three times its normal size. On section, the pulp is soft, often almost diffluent, and projects upon the cut surface. The color is sometimes dark red, sometimes grayish red, or mottled red and gray. The trabeculæ and glomeruli are usually concealed by the swollen and softened pulp, but the glomeruli are sometimes unusually prominent.

Microscopical examination shows the marked increase in size to be due in part to the hyperæmia; in part to a swelling and increase in the number of cells, sometimes of the pulp, sometimes of the glomeruli, or of both. We find large, multinucleated cells; cells resembling the ovoidal and polyhedral cells of the pulp, but larger and with evident division of the nuclei. Cells resembling leucocytes may be present in large numbers, and larger and smaller cells in a condition of fatty degeneration, or containing pigment, are often seen. The elongated cells lining the cavernous veins may be swollen or increased in number. Not infrequently larger and smaller cells are found which contain structures looking somewhat like red blood-cells. In some cases, particularly in scarlatina, hyperplasia of the glomeruli is a prominent feature. In some cases, particularly in typhus and recurrent fevers, the cells of the glomeruli undergo marked degenerative changes, so that they may form small softened areas looking like little abscesses. Small necrotic areas, often associated with localized suppuration, are sometimes found in typhus and typhoid fever, scarlatina, etc., and may be due to infectious emboli. As the primary disease runs its course, the swelling of the spleen subsides, the capsule appears wrinkled, the color becomes lighter, and sometimes the organ remains for a long time, or permanently, small and soft.

The cause of these marked changes in the spleen in infectious diseases is not understood. It seems probable that they are due to the lodgment in the organ of some deleterious materials which have found access to the blood. Whether these materials are bacteria, or products of the life processes of bacteria, or something entirely apart from these, we do not know. Bacteria have, indeed, in many cases been found in the organ under these conditions, but by no means with the frequency and abundance which the commonness and prominence of the lesion would lead us to expect if it were in all cases due to their presence.

Suppurative Splenitis (Splenic Abscess).—Small abscesses may found in the spleen as the result of minute infectious emboli, and the may coalesce to form larger abscesses; but larger and smaller abscess may form in the spleen without evidence of their embolic origi Sometimes the entire parenchyma is converted into a soft, necrotic, pur lent mass surrounded by the capsule. It is rare for simple infarctio to result in abscess, but it does occasionally occur. Abscess of t spleen may occur from the propagation of a suppurative inflammati to the organ from adjacent parts; from perinephritic abscesses, ulc and carcinoma of the stomach, etc. Abscesses of the spleen may op into the peritoneal cavity, inducing fatal peritonitis, or, owing to adhesive inflammation, the opening may occur into the post-peritone tissue, into the pleural cavity, lung, stomach, intestines, or it may op on the surface. On the other hand, the contents of the abscess m dry, shrink, and become incapsulated and calcified. Abscesses m occur in ulcerative endocarditis. pyæmia, typhoid fever, and more rare in intermittent fever, and under a variety of other conditions who nature is unknown to us.

Chronic Indurative Splenitis (Chronic Splenic Tumor).—The may be, as we have already seen, a new formation of connective tissue the spleen as a result of chronic congestion or infarctions, or abo abscesses. But there is a more diffuse formation of connective tissu usually in the nature of an hyperplasia, which occurs under a variety conditions, and is now marked and extensive, and again comparativel ill-defined. It is always associated with more or less extensive chang in the parenchyma. In its most marked form it is found in chron malarial poisoning, and under these conditions it may be found not onl in persons who have suffered from repeated attacks of intermittent feve but also in those who have not thus suffered but have resided in mal rial regions. The enlarged spleen is often called "ague cake." Sim lar conditions, though usually less marked, may occur in congenital an acquired syphilis, from prolonged typhoid fever, and as a result of acut hyperplastic splenitis from various causes, and also in leukæmia an pseudo-leukæmia.

The gross appearance of the spleen in chronic indurative spleniti varies greatly, both in the size of the organ and in the appearance of th section. The spleen may be enormously enlarged or it may be of abou normal size. It is usually, however, enlarged. The capsule is usuall more or less thickened, frequently unevenly so. The consistence usually considerably increased, but this is not always the case. Th color and appearance of the cut surface present much variation. It ma be nearly normal or it may be grayish, or dark brown, or nearly black The color may be uniform or the surface may be mottled. The glomerul may be scarcely visible or very prominent; the trabeculæ are in som cases nearly concealed by the pulp; in others they are large, prominent, and abundant, so that the surface is crossed in all directions by an interlacing network of broader and narrower irregular bands, between which the red or brown or blackish pulp lies.

Not less varied are the microscopical appearances of the spleen under these conditions. In one class of cases, there is more or less uniform hyperplasia of both pulp and interstitial tissue. The parenchyma cells are increased in size and number; there may be swelling and proliferation of the lining cells of the cavernous veins (see Fig. 172). The reticulum of the pulp, as well as that of the glomeruli, and also the trabeculæ, are thickened. In another class of cases, the thickening of the reticular and trabecular tissue, either uniformly or in patches, is the prominent feature (Fig. 173), while the changes in the pulp are rather secondary and atrophic. In both forms, irregular pigmentation is frequent, the pigment particles being deposited either in the cells of the pulp or glomeruli, or in the new-formed interstitial tissue (Fig. 174). Finally, there are all intermediate forms of induration between those described, and the changes are by no means uniform in the same organ. When these spleens are large, they are liable to displacement.

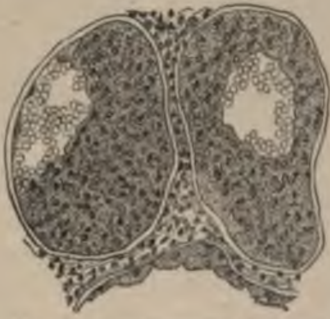

FIG. 172.—CHRONIC INDURATIVE SPLENITIS.
Showing swelling or proliferation of the lining cells of the cavernous veins.

Syphilitic Splenitis.—This lesion may present itself as an indurative process due to the formation of new connective tissue, and present no distinct morphological characteristics. In rare cases, however, gummata may be present in connection with the new fibrous tissue; then the nature of the lesion becomes evident.

Tubercular Splenitis.—This lesion is secondary, either to tubercular inflammation in some other part of the body, or is the result of the general infection in acute general miliary tuberculosis. The tubercles may be very numerous and still invisible to the naked eye, or they may be just visible, or as large as a pin's head or thereabouts, and very thickly strewn through the organ or sparsely scattered. In other cases, tubercles are larger, sometimes as large as a pea, and they are then usu not very numerous. Microscopically they present the usual variety

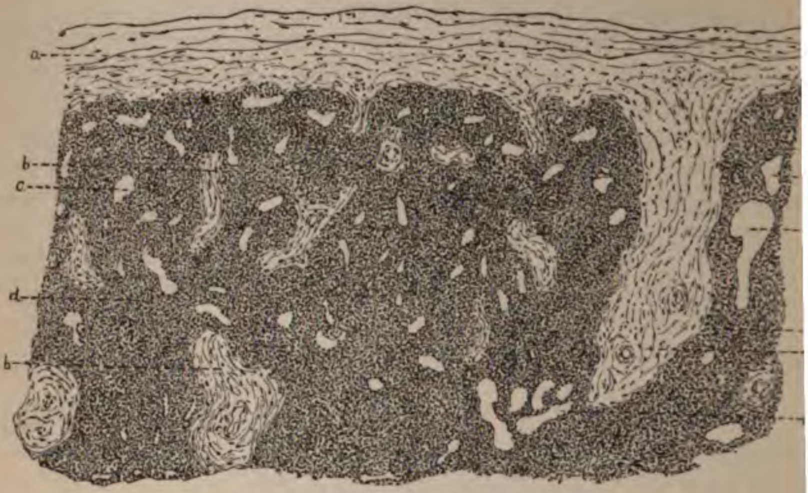

FIG. 173.—CHRONIC INTERSTITIAL SPLENITIS.

a, thickened capsule; *b*, thickened trabeculæ; *c*, dilated cavernous veins; *d*, dense pulp tis with obliterated cavernous veins.

structure: sometimes as simple tubercle granula, sometimes as cong merate tubercles; they may consist simply of a collection of small spl roidal cells, or there may be larger polyhedral cells and giant cells, wi

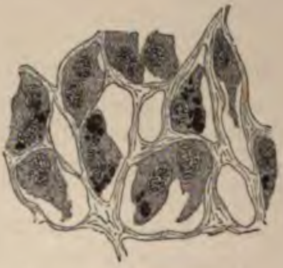

FIG. 174.—MALARIAL SPLEEN.

Showing thickening of the trabecular network of the pulp, with pigmentation of the pulp cells

a well-defined reticulum. Cheesy degeneration occurs under the usu conditions. Tubercle bacilli are usually present, particularly in the mo acute forms, sometimes in small, sometimes in enormous number They seem to be especially abundant in acute general miliary tubercul sis of children. These tubercles may be formed in the glomeruli, in th walls of the smaller arteries, in the pulp tissue, and in the trabeculæ an capsule. Owing to the peculiar character of the spleen tissue, the earlier stages are not readily recognized, since simple collections of small spheroidal cells are not distinctly outlined against the normal tissue. There is frequently a moderate swelling of the spleen, owing to hyperæmia and hyperplasia of the parenchyma.

Perisplenitis.—Acute inflammation of the capsule of the spleen may occur as a part of a general or localized peritonitis, or as a result of lesions of the spleen itself, such as infarctions, abscesses, and acute hyperplastic inflammation. Under these conditions, a fibrinous pellicle, with more or less pus, may be formed on the surface of the organ. *Chronic perisplenitis,* resulting in the production of new connective tissue, either in patches or as a more or less general thickening of the capsule, is of frequent occurrence. It may follow acute inflammation of the capsule, or be a part of general or localized chronic peritonitis. It is common in connection with chronic indurative splenitis, and it may occur from unknown causes. Sometimes the capsule is three or four mm. in thickness over a considerable area; sometimes very small nodular thickenings or papillary projections occur. As a result of this process, adhesions, sometimes very extensive, may form between the spleen and adjacent parts. The thickened capsule is sometimes more or less extensively calcified.

Alterations of the Spleen in Leukæmia and Pseudo-Leukæmia.—The lesions of the spleen are essentially the same under both of these conditions. They consist, in general, of an hyperplasia, sometimes most marked in one, sometimes in another of the structural elements of the organ, but usually they all participate in the alterations. The changes which occur in the earlier stages are but little known. The gross appearances of the spleen, as we find them in persons dying of either of the above diseases, present considerable variation. They are usually enlarged, and sometimes are ten or fifteen times the normal size. They are usually hard, but are sometimes of the ordinary consistence, or softer. The capsule is usually thickened and rough. The section of the spleen may be of a uniform dark-red color, but it is more frequently mottled red and gray. Sometimes the glomeruli are inconspicuous, but they are very often enlarged and prominent. They may be two to four mm. in diameter, and, owing to an infiltration of the arterial sheaths with lymph cells, may appear to the naked eye as grayish, round or elongated bodies, arranged along branching interrupted grayish streaks. The trabecula may be greatly thickened, as also the reticulum of the pulp, so as to be evident to the naked eye. Brown or black pigment may be collected around the glomeruli or in the pulp. Hæmorrhagic infarctions or circumscribed extravasations of blood may further complicate the picture.

Microscopically the appearances are essentially the same as those above described in acute hyperplasia and in chronic interstitial splenitis, depending upon the stage and variety of the disease. Owing to the g size which some of these spleens attain, they are liable to displacen and they may interfere by pressure with the functions of neighbo organs.

DEGENERATIVE CHANGES IN THE SPLEEN.

Atrophy.—The spleen may become atrophied in old age; as a re of prolonged cachexiæ, and in connection with profound and persis anæmia; or, more rarely, from unknown causes. The capsule ma wrinkled and thickened, the color pale, the trabecula prominent, consistency increased. The change is largely in the pulp, whose pa chyma cells are decreased in number.

Amyloid Degeneration.—This degeneration may affect the glome or the pulp tissue, or both together. When confined to the glome the spleen may or may not be enlarged, and the cut surface is mor less abundantly sprinkled with round or elongated translucent bo resembling considerably in general appearance the grains of boiled s These are the waxy glomeruli. Such spleens are often called "s spleens." Microscopical examination shows that the degeneratio confined to the walls of the arteries, capillaries, and reticulum of glomeruli, with atrophy and disappearance of the lymphoid cells.

In other cases, either with or without involvement of the glomer there is waxy degeneration of the blood-vessels and reticulum of pulp, which may occur in patches or be general and more or less cessive. If the alteration is general and considerable, the splee enlarged, its edges rounded, its consistence increased. On section appears translucent, and the distribution of the degenerated areas ma readily seen by holding a thin slice up to the light. The spleen may alone affected, or there may be similar degenerations in other orga The general conditions under which this lesion occurs, and the meth of staining and studying, are given on page 66.

Pigmentation of the spleen may occur as the result of the decom sition of hæmoglobin in the organ under a great variety of conditio thus after hæmorrhagic infarctions, small multiple hæmorrhages, ac hyperplastic splenitis, etc. Or the pigment may be anthracotic and brought to the organs from the lungs or bronchial glands (see page 6 Bile pigment may also be deposited in the spleen in jaundice. The p ment may lie in the walls of the smaller arteries, in the cells and retic lum of the pulp, or free in the latter tissue, or in the follicles. It usually quite unevenly distributed. The pigment may be red, brow or black. According to Weigert, anthracotic pigment may be sometim seen with the naked eye in the periphery of the glomeruli, as da crescents.

TUMORS.

Primary tumors of the spleen are rare. Small *fibromata, sarcomata,* and *cavernous angiomata* sometimes occur. *Sarcomata* and *carcinomata* may occur in the spleen secondarily either as metastatic tumors or by extension from some adjacent part, as the stomach. Dermoid cysts are described. but are rare. Other larger and smaller cysts, whose mode of origin is in most cases obscure, not infrequently occur.

PARASITES.

Pentastomum denticulatum is not infrequently found in the spleen, usually incapsulated and calcified. *Cysticercus* is rare. *Echinococcus* is occasionally found, and, if the cysts are large or numerous, may cause more or less extensive atrophy of the organ.

Various forms of bacteria have been found in the spleen. *Micrococci* have been found in pyæmia, small pox, ulcerative endocarditis, diphtheria, and under other conditions. The *Bacillus anthracis* occurs here in anthrax; the *B. tuberculosis* in tubercular inflammation; and bacilli have been described in typhoid fever. *Spirochæte Obermeieri* may be found in relapsing fever.

MALFORMATIONS AND DISPLACEMENTS.

The spleen may be absent in acephalous monsters, and with defective development of other abdominal viscera. Very rarely it is absent in persons who are otherwise perfectly developed. Small accessory spleens from the size of a hazelnut to that of a walnut are not infrequent. They usually lie close to the spleen, but may be considerably removed from it; thus they have been found imbedded in the head of the pancreas. Two spleens of about equal size have been observed. The form of the spleen is subject to considerable variation. It may be made up of several distinct lobes. It may be displaced congenitally or as the result of disease. It may be on the right side in transposition of the viscera. As the result of congenital defects in the diaphragm, the spleen may be found in the thorax; or in deficient closure of the abdominal wall it may, together with other abdominal viscera, be found outside of the body.

The spleen may be pressed downward by any increase in the contents of the thorax. It may be fastened by adhesions to the concave surface of the diaphragm, so that its long axis is nearly horizontal instead of vertical. It may be displaced by changes in the contents of the abdominal cavity. If the organ is increased in size, it frequently becomes tilted, so that its lower border reaches the right iliac region. If the ligaments are too long congenitally, or if they are lengthened by traction, and if the organ is at the same time increased in weight, it become very movable. It may sink downward with its hilus t upward; or it may be rotated on its axis, and, owing to torsion o vessels thus produced, the organ may atrophy; or the pressure o ligaments and vessels across the duodenum may cause occlusion c gut.

THE PANCREAS.

The diseases of the pancreas are, so far as we know, with a few exceptions, of no great practical importance; that is, they do not often give rise to symptoms of disease or cause death, but the lesions are found in the bodies of persons dead from other diseases. It is probable, however, that in many cases their apparent insignificance is due to our lack of knowledge of the interference with functions which lesions of the gland induce, and to the incomplete examination of the pancreas which is so common at autopsies.

Hæmorrhage into the substance of the pancreas may occur as the result of injury; in the hæmorrhagic diathesis; in connection with valvular diseases of the heart or interference with the portal circulation; or in connection with extensive fatty degeneration and fat necrosis of the organ. Such hæmorrhages may be minute or extensive. Several cases of sudden death are recorded in which the only discoverable lesion was an extensive hæmorrhage into the substance of the gland and the tissue about it. In these cases it has been assumed that death was caused by interference with the heart's action, through pressure on the solar plexus and semilunar ganglion. but it may be due to other causes (see below, Fat Necrosis). The hæmorrhage may be moderate and limited to the pancreas. or it may extend into the subperitoneal tissue for a considerable distance.

Hæmorrhage of the pancreas may be associated with acute inflammatory changes and with more or less extensive *gangrene* of the organ. The gangrenous pancreas may be more or less incapsulated; it may lie, bathed in pus, in the abdominal cavity; it may, by ulceration of the intestinal wall, get into the gut and be discharged with other intestinal contents.

INFLAMMATION.

In some cases of typhoid fever, pyæmia, yellow fever, and other acute infectious diseases, the pancreas is red, swollen, and œdematous. Microscopically the most prominent lesion is a swelling and undue granulation of the glandular epithelium, and hyperæmia. This condition is known as *Parenchymatous Pancreatitis.*

Suppurative Pancreatitis is not very common, and may be prim or due to the extension of a suppurative inflammation from adjacen distant parts of the body. There may be a diffuse infiltration of organ, with pus cells or larger and smaller abscesses. The abscesses open into the gastro-intestinal canal or into the peritoneal cavity. causes of primary suppurative pancreatitis are often most obscure. may be associated with fat necrosis and with hæmorrhage and gangr of the pancreas.

Chronic Interstitial Pancreatitis (Cirrhosis of the Pancreas).—T lesion consists in an increase of interstitial connective tissue, which be general or confined to some particular portion of the gland. organ is sometimes enlarged, sometimes smaller than normal. I usually dense and hard; secondary atrophy of the parenchyma regul occurs. It may be due to chronic inflammatory processes in the vicin of the organ.

Syphilitic Inflammation.—Chronic interstitial pancreatitis is quently found in congenital syphilis of the new-born, and the gross microscopical lesions are similar to those above described. It is not d nitely established whether or not a similar lesion may be caused acquired syphilis. Gummata are very rare in the pancreas, but h been described in congenital syphilis in very young children.

Tubercular Inflammation.—Larger and smaller tubercles and tub cular, cheesy nodules are occasionally found in the pancreas in connecti with acute general miliary tuberculosis or with tubercular inflammati in some other organ, particularly with that of adjacent lymph nod the lungs, and the intestine.

DEGENERATIVE CHANGES IN THE PANCREAS.

Atrophy of the pancreas may occur in old age, and as a result pressure from tumors or other adjacent structures. Marked atrophy the pancreas is found in a certain proportion of cases of diabetes m litus, but it is not constant.

Fatty Degeneration of the parenchyma cells may occur, and in son cases is so extensive as to lead to nearly complete destruction of the protoplasm.

Fatty Infiltration, which should be distinguished from fatty degen ration, consists in the accumulation of fat in the interstitial tissue of t gland. This may be so excessive as to cause nearly entire destruction the gland structures. Under these conditions, the outline of the org may be preserved. the fat being inclosed by the capsule.

Amyloid Degeneration.—This usually occurs in connection wit similar degeneration in other organs, and is confined to the walls of th blood-vessels and the interstitial tissue.

Fat Necrosis.—A very peculiar lesion of the fat tissue, most fr quently seen in the fat tissue about the pancreas or between its lobules, but sometimes in fat tissue in other parts of the body, has been a few times described and called *fat necrosis*. White or yellowish nodules, varying from the size of a pin's head to that of a pea or larger, are seen imbedded in the fat, the central portion being often soft and grumous and readily squeezed out. They are sometimes calcified and sometimes surrounded by a connective-tissue capsule. Microscopical examination shows degeneration and disintegration of the fat tissue (Fig. 175). They are most frequently found in marasmatic persons. When the lesion is extensive, according to Balser, it may cause death either directly or by inducing hæmorrhage. Some of the extensive hæmorrhages about the pancreas, above mentioned, may be caused in this way.[1]

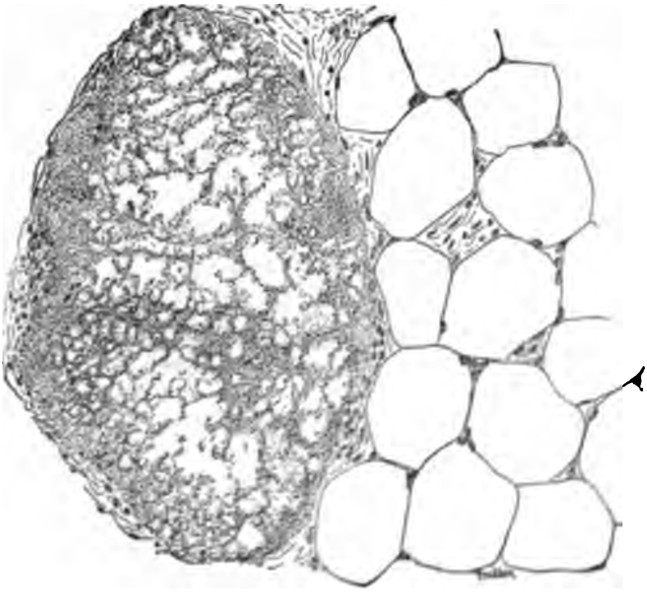

FIG. 175.—FAT NECROSIS IN THE PANCREAS.

Drawn from a specimen prepared by Dr. Ira Van Gieson, and reported to the New York Pathological Society, 1888.

TUMORS.

Carcinomata are the most common and important of the tumors of the pancreas. They may be primary or secondary. Primary carcinomata are most frequently found in the head of the organ, but may occur in other parts. The hard or scirrhous form is most common, but o sionally soft and succulent and colloid forms are found. They liable to involve adjacent parts by continuous growth, and may f metastases in the liver, adjacent lymph nodes, etc. Secondary carcin in the pancreas may occur in carcinoma of the stomach, duodenum, the gall ducts and gall bladder. As a result of carcinoma of the pancr aside from the extension of the growth, there may be pressure on ductus choledochus, with jaundice; or on the pancreatic duct, with cy dilatation; or pressure on the duodenum, with stenosis of the gut; pressure on the vena cava, or portal vein, or superior mesenteric v etc., with disturbances of the circulation.

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[1] For a detailed consideration of acute inflammation, hæmorrhage, gangrene, and fat necrosis of the pancreas, with bibliography, consult Fitz, Middleton Goldsmith Lecture for 1889 on "Acute Pancreatitis," Transactions New York Pathological Society, 1889.

Concretions of carbonate and phosphate of lime are frequently fou in the pancreatic ducts. They are usually multiple, small, whiti smooth, or of rough and irregular shape. Sometimes, however, t reach a diameter of more than an inch. They consist chiefly of calci phosphate and carbonate. Besides these free concretions, the walls the ducts are sometimes incrusted with salts of lime. Such concreti may produce dilatation of the pancreatic ducts and large cysts, or m rarely abscesses.

Foreign Bodies.—Gall stones sometimes find their way into the p creatic duct. Ascarides have been found in the ducts in a considera number of cases.

Dilatation of the pancreatic ducts and the formation of cysts ta place in several different ways.

1. The entire duct may undergo a uniform cylindrical dilatatio With this cylindrical dilatation we sometimes find associated small s culi.

2. There may be sacculated dilatations at some point in the duc These dilatations form cysts of large size, as large even as a child's hea Their walls frequently undergo degeneration and calcification. The cysts often become filled with blood, and may then be mistaken for a eurisms.

3. The small branches of the pancreatic duct may be dilated so to form a number of small cysts. These cysts are filled with seru mucus, pus, or a thick, cheesy material.

MALFORMATIONS AND DISPLACEMENTS.

The pancreas may be entirely absent in anencephalous and doub monsters, and in congenital umbilical herniæ. The pancreatic duct ma be double; it may open into the duodenum at some distance from th biliary duct, or into the stomach. The head of the pancreas may be u duly developed, and sometimes even completely separated from the res of the organ, opening into the duodenum with a duct of its own. Occa sionally there is a small accessory pancreas situated beneath the serosa of the duodenum or stomach.

The pancreas is so firmly bound down that its position is not often changed. Sometimes, however, it is found pressed downward by tight lacing, displaced by aneurisms, or contained in umbilical and diaphragmatic herniæ.

THE SALIVARY GLANDS.

THE PAROTID, SUBMAXILLARY, AND SUBLINGUAL.

INFLAMMATION.

This condition is most frequent and important in the parotid.
epidemic disease known as *mumps* is most frequently confined to
parotid gland of one side, but the submaxillary and sublingual ma
at the same time involved. The gland is swollen and there is o
œdema of the mucous membrane of the mouth and pharynx.
little is known of the actual minute changes which the gland under
in this disease.

Acute parotiditis occasionally occurs as a secondary lesion in a var
of diseases, as in typhoid and scarlet fever, pyæmia, pneumonia,
and by propagation of inflammation from the mouth. Under these
ditions the inflammation is usually suppurative, and frequently res
in abscess or sloughing. The interstitial tissue of the gland is mor
less densely infiltrated with pus cells, and the parenchyma cells
undergo fatty degeneration and disintegration. The inflammation
be confined to the gland or it may spread to adjacent parts, someti
causing much destruction of tissue, and may give rise to inflammat
of the brain or of the inner ear, or even to metastatic pyæmic absce
in different parts of the body. Healing may occur, with the format
of salivary fistulæ.

The submaxillary gland may be involved with the parotid in the s
purative inflammation.

Acute suppurative inflammation of the connective tissue about
submaxillary gland is sometimes of serious import. Sloughing and g
grene may occur, and are apt to spread to adjacent parts. Septicæm
œdema of the glottis, or pneumonia may complicate the process a
cause death.

The sublingual gland is not often the seat of inflammation.

Chronic inflammation, leading to the formation of dense interstit
tissue, sometimes occurs in the salivary glands. This may occur by
self or follow an acute inflammation.

The *Excretory Ducts* of the salivary glands may become inflamed from the presence of foreign bodies or of concretions formed in them. They may become occluded from the presence of calculi or as the result of inflammation, and may thus become widely dilated both in the main branches and in the finer ramifications. The dilatation of Wharton's duct to form larger and smaller cysts containing salivary fluid, sometimes gives rise to very large and troublesome tumors which constitute one of the forms of *ranula*.

TUMORS.

Fibromata are of occasional occurrence in the parotid. *Chondromata*, *sarcomata* and *fibro-sarcomata*, and *myxomata*, or more frequently mixed tumors formed of varied combinations of these, are of frequent occurrence in the parotid, and of occasional occurrence in the submaxillary gland. These complex or mixed tumors are of more frequent occurrence in these glands than in any other part of the body, except possibly the ovary. They are sometimes rendered still more complicated in structure by a partially atypical glandular growth lending them an adenomatous character, and by the formation of cysts. Or they may present in parts a distinctly carcinomatous character.

Fibro-sarcoma and *melano-sarcoma* have been described. *Primary carcinoma* of these glands is very rare.

One case of *rhabdomyoma* of the parotid gland, with evidences of atypical development of portions of the gland, has been described by one of us.[1]

PARASITES.

Echinococcus has been observed in the parotid gland.

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[1] *Prudden*, "Rhabdomyoma of the Parotid Gland" American Journal of the Medical Sciences, April, 1883.

THE THYROID GLAND.

Hyperæmia of the thyroid gland, often accompanied by considera enlargement of the organ, may be the result of valvular disease of t heart; it occurs in Basedow's disease; it may be temporary or pern nent, and in the latter case may give rise to the formation of new c nective tissue. Hæmorrhages may occur, causing pigmentation of t organ.

Inflammation of the thyroid gland is not very common, and m occur from a variety of causes. It may result in the formation of larg and smaller abscesses, or in the production of new connective tiss *Tubercular inflammation*, with the formation of miliary tubercles, is infrequent occurrence. *Syphilitic inflammation*, with the formation gummata, has been described, but is rare.

Degeneration.—Colloid degeneration of the epithelial cells of t gland, and the filling of the alveoli with colloid material, is of comm occurrence, and when occurring in moderate degree may be regarded a normal event, since a certain amount of this change is found in ma otherwise apparently normal glands. It may occur, however, to such extent as to constitute a lesion (see below).

Amyloid degeneration, particularly of the blood-vessels, is of infr quent occurrence.

TUMORS.

Among the most important of the lesions of the thyroid is the enlarg ment of the organ commonly known as the *goître* or *struma*. T enlargement of the gland may occur in several different ways, and i only a part of the cases is to be considered as a tumor. Thus, a simp hyperæmia may, as above stated, cause considerable enlargement of t organ, and this is sometimes called *Struma hyperæmica*. The true goîtr however, consists in the enlargement of the old and the formation new gland alveoli, while with these changes there is very frequently ass ciated a greater or less amount of colloid degeneration. When there i new formation of gland tissue, the growth has the character of an *ad noma*. The hyperplasia may occur diffusely, so that the whole gland i more or less enlarged: or it may occur in the form of circumscribe nodules. When the colloid degeneration is prominent, so that th tumor has a gelatinous look, it is called *colloid struma*. Accumulations of fluid, blood, colloid, etc., in the old or new-formed alveoli, may cause dilatation and atrophy of the walls of the alveoli, so that cysts, sometimes of large size, are formed. Thus occurs the *cystic struma*. Again, the blood-vessels may undergo marked dilatation, so that we may have a *telangiectatic struma ;* or *cavernous angiomata* may form within them. Very frequently all these varieties of lesions are present in the same goître. The appearances may be rendered still more complex by the occurrence of hæmorrhages and pigmentation, calcification, purulent or indurative inflammation, and by the not very infrequent association with carcinoma and sarcoma. The cause of goître is not well understood. The growth is, as a rule, slow, but occasionally a very rapid enlargement occurs as the result of a sudden increase of the colloid degeneration. In many cases, even very large goîtres give rise to but moderate inconvenience, but they may assume great significance by incroaching upon neighboring parts. Thus death may be caused by pressure on the trachea, œsophagus, or on the large vessels.

Sarcoma, either spheroidal or spindle-celled, may occur as primary tumors in the thyroid, either in otherwise normal glands or in connection with struma. *Melano-sarcoma* has been observed. Secondary sarcomata are rare.

Primary *carcinoma*, both glandular and scirrhous, occurs in the thyroid, and, particularly in the softer forms, may spread to adjacent parts, and occasionally form distant metastases.

PARASITES.

Echinococcus cysts have been found in the thyroid.

The thyroid gland is sometimes very small, either as the result of atrophy or as a congenital deficiency. This is most marked in the condition called *myxœdema* (see below).

MYXŒDEMA.

This disease occurs most frequently in middle-aged women, and its cause is unknown. The skin of the face is apt to be swollen and waxy, causing a peculiar and rather characteristic appearance of the features. The skin of the body is apt to be dry and rough, and the hair may fall out. Perspiration is, as a rule, diminished. The mental condition is dull, and loss of memory and insanity may occur. Bodily movement and speech are apt to be impaired.

The fat tissues may be atrophic, and the subcutaneous tissue has been shown in some, though not all, of the cases to contain an unusual amount of mucin. In some cases, the fibres of the upper layers of the corium are crowded apart by fluid.

The most marked and constant lesion in this disease is an atrophi condition of the thyroid gland. The parenchyma of the gland is mor or less completely replaced by fibrillar connective tissue and by new formed reticular tissue resembling the lymphatic tissue of the lymp nodes.

The general appearance of the atrophied thyroid gland is shown i Fig. 176.

In a case reported by Hun, which one of us has examined, the lobe of the thyroid measured less than one-half of an inch in diameter, an the entire gland weighed only about 7.2 gm. (112 grains).

In addition to the lesion of the thyroid, there are apt to be chroni endarteritis and chronic diffuse nephritis. In some cases there is a accumulation of small spheroidal cells about the smaller blood-vessels i various parts of the body, and also petechial hæmorrhages.

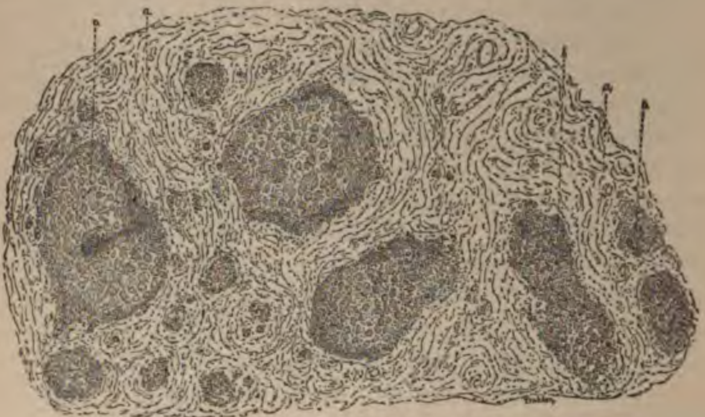

FIG. 176.—SECTION OF THE ATROPHIED THYROID GLAND IN MYXŒDEMA.

a, interstitial tissue; *b*, atrophied lobules with small spheroidal-celled or lymph tissue in their peripheries.

While the atrophy of the thyroid is the most marked and frequent lesion in this disease, our lack of knowledge about the function of this gland prevents a definite conception as to the relationship of this change to the symptoms.

By the destruction of the thyroid from disease, or as the result of its removal in men and animals, a condition considerably resembling myxœdema is apt to be induced.

Myxœdema appears to be identical with that condition which has been described as *cachexia strumipriva*.[1]

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[1] For detailed descriptions of myxœdema, and the literature, see *Hun and Prudden*, "Myxœdema," Am. Jour. Med. Sciences, July and August, 1888, and "Report on Myxœdema" in Supplement to vol. xxi. Clinical Society Transactions, London, 1888.

THYMUS GLAND.

The thymus occasionally but not usually persists until youth or middle age instead of undergoing the usual developmental atrophy.

Small *hæmorrhages* are described in the thymus of young children as the result of venous congestion in asphyxia, etc. They may also occur in the hæmorrhagic diathesis.

Suppurative Inflammation of the thymus is of occasional occurrence, and is usually secondary to a similar inflammatory process in some other part of the body.

Tubercular and syphilitic inflammation of the thymus are described. The sarcomata are the most common tumors of the thymus.

THE SUPRARENAL CAPSULES.

MALFORMATIONS.

In acephalic and other monsters, the suprarenal capsules may atrophied or entirely absent. Sometimes in well-formed adults th organs cannot be discovered.

There may be little rounded nodules loosely attached to the surf of the capsules and having the same structure.

If one of the kidneys is absent or in an abnormal position, its sup renal capsule usually retains its proper position.

HÆMORRHAGE.

In children, soon after birth, it is not very infrequent to find lar hæmorrhages in one of the capsules, converting it into a cyst filled wi blood. The same lesion has been observed in a few cases in adults.

THROMBOSIS.

Klebs describes a case of capillary thrombosis of the cortex in bo capsules in a woman after excision of the knee joint.

INFLAMMATION.

The most frequent lesion of the suprarenal capsules is *tubercular i flammation*. They are usually increased in size, their surfaces are smoot or nodular. The normal structure of the gland is lost, and is replace by tubercle tissue, connective tissue, and cheesy matter (see Addison Disease).

Suppurative Inflammation, with the formation of abscesses, has bee seen in a few cases.

DEGENERATION.

Fatty Degeneration of the cortical portion of the capsules is the rul in the adult. It sometimes occurs in nodular areas. In children unde five years of age, it is a pathological condition.

Amyloid Degeneration may involve both the cortical and medullar portions. In the cortex, it usually involves only the walls of the blood vessels; in the medulla, both the blood-vessels and the cells of the parenchyma may undergo this degeneration. The capsules are usually firm and of a grayish, semi-translucent color.

TUMORS.

Carcinoma of the suprarenal capsules is not common. It may be primary, but is much more frequently secondary. Either one or both of the capsules may be the seat of the new growth.

Sarcoma occurs as a primary and secondary growth. Probably many of the older cases described as cancers were really sarcomata.

Cylindroma.—Klebs describes a growth of this character in one of the capsules, secondary to a tumor of the same kind in the supra-orbital region. He gives to such tumors the name of lymphangioma cavernosum. The exact character of these growths is still obscure. They consist of irregular follicles and cavities, lined with epithelium, and containing peculiar hyaline, structureless bodies.

Cysts are found both single and multiple. They are usually situated in the cortex.

Neuroma.—A ganglionic neuroma has been described by Weichselbaum.[1]

Glioma has been described as occurring in the medullary region.

An hyperplasia of the gland tissue, with fatty degeneration in the form of circumscribed nodules, is described by Virchow.

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[1] Virch. Archiv, Bd. 85, p. 554

THE URINARY APPARATUS.

THE KIDNEYS.

MALFORMATIONS.

Entire absence of both kidneys is sometimes associated with gr malformation of the entire body. Such fœtuses are not viable.

Absence of one kidney is not uncommon, the left kidney being m frequently absent than the right. The absence of the kidney may complete, the ureter being also absent; or there may be a little mass connective tissue and fat to represent the kidney, and a ureter runni down to the bladder. The single kidney which is present is usua much enlarged. It may be in its natural position or displaced dow ward.

Since the extirpation of the kidney has been practised by surgeons, has been found that absence of one kidney is more common than w formerly believed.

When both kidneys are present, one of them may be much larger th the other.

Sometimes one kidney will have two pelves or two ureters.

A rather frequent malformation is the so-called *horseshoe kidne* The lower ends of the kidneys are joined together by a commissu The commissure is usually composed of kidney tissue, but sometimes connective tissue. The two kidneys may be normal, except for the cor missure; or their shape, the arrangement of the vessels and ureters, a the position, may be unnatural.

The two kidneys may be united throughout so as to look like a sing misshapen kidney with two or more pelves and irregular blood-vessel The united kidneys may be both situated on one side of the vertebr column or in the pelvis.

CHANGES IN POSITION.

The kidneys may be placed in an abnormal situation, in which the are either fixed or movable.

The change in position is either lateral or downward. When di placed downward, the kidney may be over the sacrum or below this in the cavity of the pelvis. The vessels also have an irregular origin and distribution. The kidney is firmly attached in its abnormal position.

Movable or wandering kidneys are found in adult life as a result of tight lacing, of pregnancy, of over-exertion, and of unknown causes. They are more frequent in females than in males. The right kidney is the one more frequently affected. The blood-vessels become lengthened and the attachments of the kidney longer and looser.

BRIGHT'S DISEASE.

This name is used as a convenient term to group together a certain number of diseases of the kidney. This group may be subdivided as follows:

I. Acute Bright's Disease.
  1. *Acute Congestion of the Kidney.*
  2. *Acute Degeneration of the Kidney.*
  3. *Acute Exudative Nephritis.*
  4. *Acute Diffuse Nephritis.*

II. Chronic Bright's Disease.
  1. *Chronic Congestion of the Kidney.*
  2. *Chronic Degeneration of the Kidney.*
  3. *Chronic Diffuse Nephritis with Exudation.*
  4. *Chronic Diffuse Nephritis without Exudation.*

I. Acute Bright's Disease.

1. *Acute Congestion of the Kidney.*—This change in the kidneys we know rather from clinical than from anatomical experience.

It is caused by the ingestion of some poisons, especially cantharides, by severe injuries, and by surgical operations. It also occurs as a temporary condition in the course of chronic Bright's disease.

The characteristic symptom is diminution in the quantity, or suppression, of urine.

2. *Acute Degeneration of the Kidney.*—This is an acute change in the kidneys, characterized by degeneration or death of the epithelium of the tubes and of the capsule cells of the glomeruli. It is also called "parenchymatous inflammation."

It is caused by severe inflammations of other parts of the body; by the presence in the body of the poisons of the infectious diseases; and by the ingestion of poisons, especially of arsenic, mercury, and phosphorus.

After death the gross appearance of the kidneys varies with the intensity of the morbid process and with the presence or absence of secondary changes on the part of the blood-vessels. In the milder cases the kidney is a little swollen and the cortex is pale; in the severe cases kidney is much enlarged and the cortex is either white or congested

The changes in the renal epithelial cells are: a simple swelling of cell body, especially of its network, causing it to look larger and n opaque and to take on irregular shapes; an infiltration of the cell bo with granules of albuminoid matter and fat; a death of the cells, wh may take the form of coagulation necrosis or of a disintegration crumbling of the cell bodies; a desquamation of the dead cells; a for tion of hyaline masses in the cells; a growth of new cells to take the pl of the dead epithelium. All these changes are most marked in the c voluted tubes (Fig. 177).

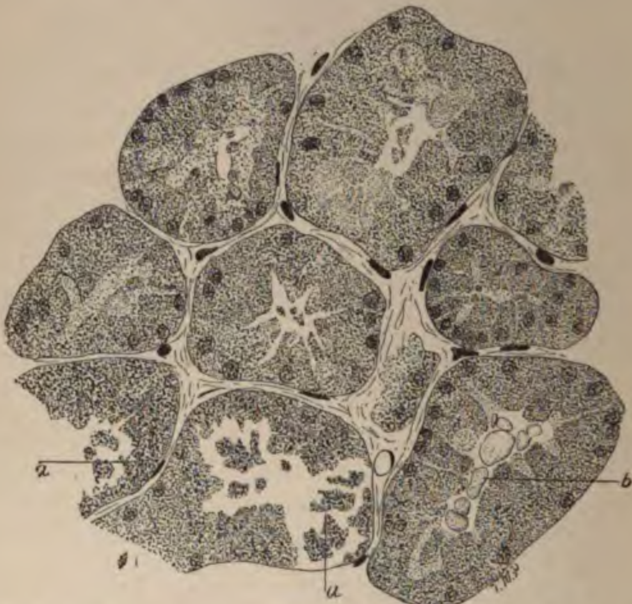

Fig. 177.—Acute Degeneration of the Kidney (Acute Parenchymatous Nephritis). From a case of yellow fever. *a*, the swollen and granular epithelium peeling off and disin grating; *b*, hyaline material in the lumen of the tubule.

If the degeneration or death of the renal epithelium is extensive ar produced quickly, it is often accompanied by congestion of the bloo vessels and exudation of serum. The congestion will be found aft death, and also casts in the straight tubules.

3. *Acute Exudative Nephritis.*—This is frequently a primary n phritis, either occurring after exposure to cold or without discoverab cause. It complicates scarlatina, measles, diphtheria, typhoid feve acute general tuberculosis, pneumonia, acute endocarditis, acute pe

tonitis, dysentery, erysipelas, diabetes, and many other of the infectious diseases and severe inflammations. It is one of the forms of nephritis which complicate the puerperal condition. It has been observed in local epidemics, apparently due to the presence of pathogenic bacteria in the kidney.

The nephritis has the ordinary characters of an exudative inflammation: congestion, an exudation of blood plasma, an emigration of white blood-cells, and a diapedesis of red blood cells; to which may be added swelling or necrosis of the renal epithelium and changes in the glomeruli.

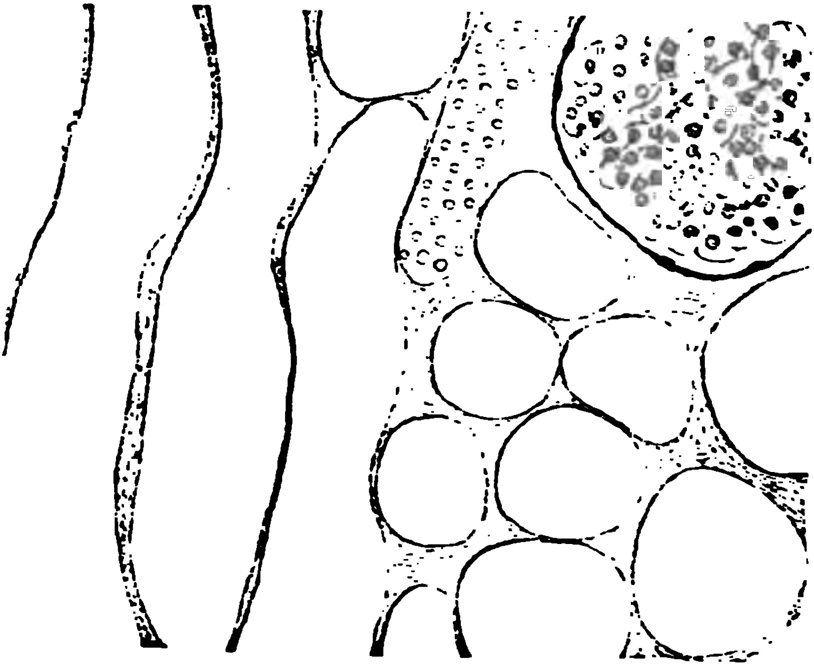

FIG. 17[illegible].—ACUTE NEPHRITIS. [illegible] Paris green [illegible] hours before death.

In the milder cases we find the inflammatory products—serum, casts, white and red blood-cells—in the urine. But in the kidneys after death we find no lesions, unless it may be a few casts in the straight tubes. The morbid process is confined to the blood-vessels of the kidney, and its only result is the exudation into the renal tubules.

In the more severe cases, we find the kidneys large and smooth, the cortex thick and white, or white mottled with red, or the entire kidney intensely congested. If the stroma is infiltrated with serum, the kidney is succulent and wet; if the number of pus-cells is very great, there will be little, whitish foci in the cortex.

There are, besides the exudation, changes in the tubes, the stro and the glomeruli. All the changes are most marked in the cort portion of the kidney.

In the tubes, the epithelium may be flattened, or swollen, opa and detached from the walls of the tubes. There may be a unifo symmetrical dilatation of all the cortex tubes. The tubes may be em or they may contain coagulated matters in the form of irregular ma and of hyaline cylinders. The irregular masses are found principall the convoluted tubes; they seem to be formed by a coagulation of

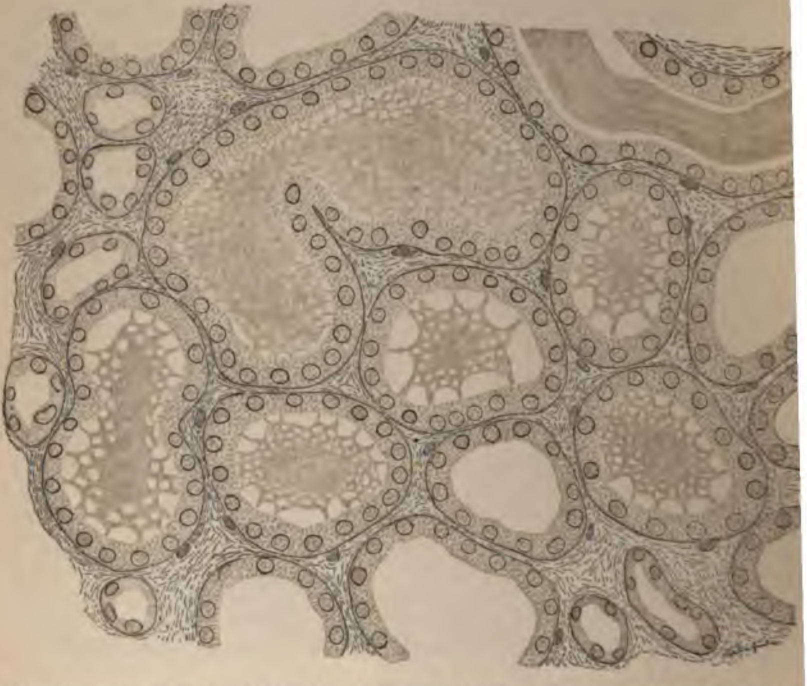

FIG. 179.—ACUTE NEPHRITIS OCCURRING WITH ACUTE GENERAL TUBERCULOSIS, × 850 and redu

stances contained in the exuded blood-plasma, and are not to be co founded with the hyaline globules so often found in normal convolut tubes.

The hyaline cylinders are more numerous in the straight tubes, b are also found in the convoluted tubes. They are also formed matter coagulated from the blood-plasma, and are identical with the cas found in the urine. The tubes may also contain red and white bloo cells.

In some cases there is an excessive emigration of white blood-cell This excessive emigration is not necessarily attended with exudatio of the blood-serum, and so the urine of these patients may contain no albumin.

The white blood-cells are not found equally diffused throughout the kidney, but are collected in foci in the cortex. These foci may be very minute or attain a considerable size. They do not resemble the suppurating foci seen with embolism or with pyelo-nephritis.

In the glomeruli we find considerable changes. The cavities of the capsules may contain coagulated matter and white and red blood-cells, just as do the tubes. The capsular epithelium may be swollen, sometimes so much so as to resemble the tubular epithelium, and this change is most marked in the capsular epithelium near the entrance of the tubes.

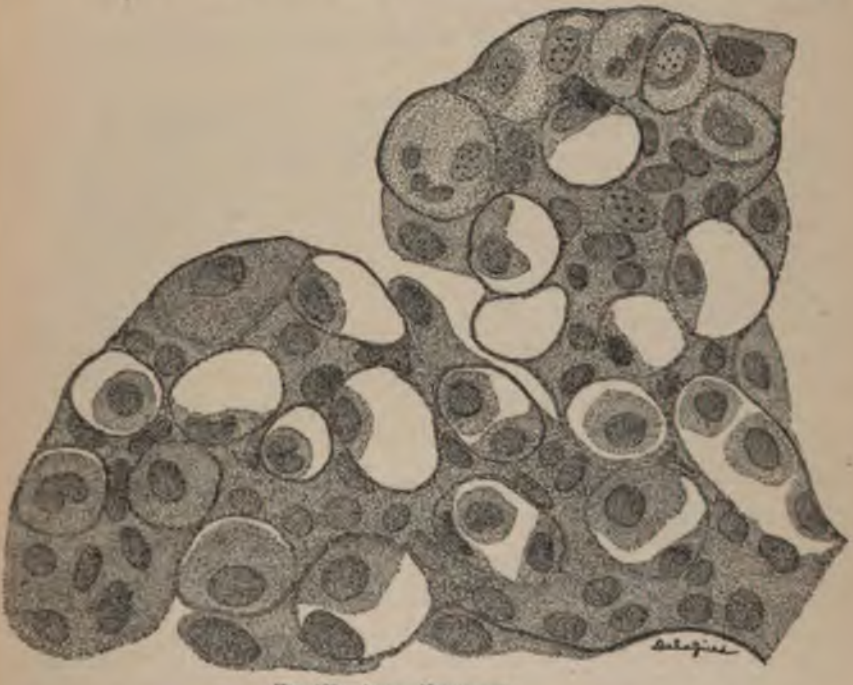

FIG. 180.—ACUTE NEPHRITIS.

Showing the swelling and growth of cells in and on the capillaries of a glomerulus in a case of scarlatina.

The most noticeable change, however, is in the capillary tufts of the glomeruli. These capillaries are normally covered on their outer surfaces by flat, nucleated cells, so that the tuft is not made up of naked capillaries, but each separate capillary throughout its entire length is covered over with these cells. There are also flat cells which line the inner surfaces of the capillaries, but not continuously, as is the case with capillaries in other parts of the body.

In exudative nephritis, the swelling and growth of cells on and in t capillaries change the appearance of the glomeruli. They are larg more opaque; the outlines of the main divisions of the tufts are visib but those of the individual capillaries are lost. This change in t appearance of the glomeruli is due to the swelling and growth of t cells on and in the capillaries (Fig. 180).

In very severe cases, the growth of the cells on the tufts is so co siderable that they form large masses of cells between the glomerul and its capsule.

The walls of the arteries in the kidneys may be thickened by swelling of their muscular coats.

Acute exudative nephritis is regularly a transitory lesion. It ma indeed, be so severe as to destroy life in a short time. But, as a rule, if the patients do recover from it, they recover completely, and the kidneys return to their natural condition.

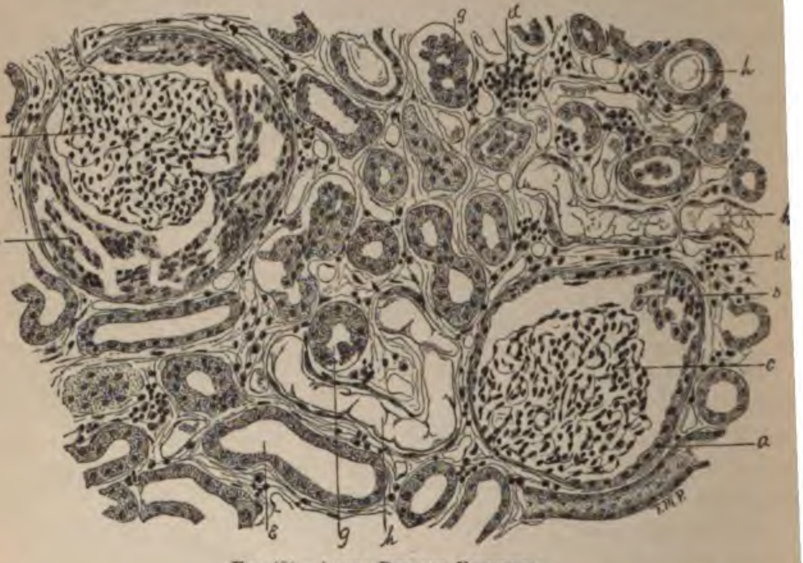

FIG. 181.—ACUTE DIFFUSE NEPHRITIS.

From a case of scarlatina. *a*, swollen capsular epithelium; *b*, proliferation of tuft cells; *c*, compressed tuft; *d*, swollen stroma infiltrated with cells; *e*, dilated convoluted tubules; *g*, swollen epithelium peeling off; *h*, hyaline casts.

4. *Acute Diffuse Nephritis.*—This is the most serious and important of all the forms of acute nephritis, not only for the reason that it involves so much of the structure of the kidney, but because its lesions are from the first of a permanent character, and because disturbances of the general circulation are so frequently associated with it.

It is one of the forms of scarlatinal nephritis; it occurs early and late in the course of diphtheria; it is the most important variety of the nephritis of pregnancy, and it is especially frequent as a primary nephritis, with or without a history of exposure to cold. The course of the inflammation is acute or subacute. If it does not prove fatal as an acute inflammation, it regularly continues as a chronic nephritis.

The changes in the kidneys are extensive and well marked.

The kidneys are large, at first smooth, later sometimes a little roughened; the cortical portion is thick, white, or mottled with yellow or red, or congested; the pyramids are red.

In these kidneys we find the same lesions as have been described as belonging to exudative nephritis, but with two additional changes—changes which are found in the earliest stages of the inflammation, and which give the characteristic stamp to the lesion: first, a growth of connective tissue in the stroma; second, a growth of the capsule cells of the glomeruli.

These changes do not involve the whole of the kidney, but symmetrical strips or wedges in the cortex which follow the line of the arteries. These wedges are small or large, few or numerous, regular or irregular, in the different kidneys. But in every wedge we find the same general characters: one or more arteries, of which the walls are thickened; glomeruli belonging to these arteries, with a large growth of capsule cells compressing the tufts; a growth of new connective tissue in the stroma around and parallel to the arteries. Between the wedges we find at first only the changes of exudative nephritis; later, a diffuse growth of connective tissue (Fig. 181).

If the nephritis is of acute type and recent, the new tissue between the tubes consists largely of cells; if the nephritis is of subacute type and longer duration, the tissue is denser and has more basement substance. Where the growth of new tissue is abundant, the tubes become small and atrophied.

The exudation from the blood-vessels is very considerable, so that the urine contains large quantities of albumin, many casts, and red and white blood-cells.

II. Chronic Bright's Disease.

1. *Chronic Congestion of the Kidney.*—This change in the kidney may be produced by any causes which interfere with the circulation of the blood and produce venous congestion. The most common cause is valvular disease of the heart.

The kidneys are of medium size or large. Their weight is increased; they are hard and uniformly congested; their surfaces are smooth. The epithelium of the cortex tubes is opaque, flattened, or swollen. The glomeruli show a dilatation of the capillaries, with more or less thickening of their walls, and swelling of the cells which cover their walls. In the stroma there is nothing but some exaggeration of the subcapsular areas of connective tissue which are found in normal kidneys. T arteries are normal, the pyramid veins are congested and sometim dilated.

The effect of this lesion of the kidney is simply to diminish the qua tity of the urine, its quality remaining good.

2. *Chronic Degeneration of the Kidney.*—This condition is produc by long-continued disturbances of the circulation due to heart disea empyema, and similar conditions, and to causes which impair the ge eral health, such as alcoholism.

The kidneys are, as a rule, considerably increased in size, weighi together from sixteen to twenty ounces; but occasionally their size much diminished. Their surfaces are smooth; the cortical portion thickened, of pink or white color; the pyramids are red.

The epithelium of the cortex tubes is swollen, coarsely granular, infiltrated with fat. If the kidney lesion is due to disturbances of t circulation, the capillaries of the glomeruli are dilated. There are changes in the stroma or arteries. At times there is a small exudati from the blood-vessels, and casts and albumin appear in the urine.

These changes in the kidney do not, as we might expect, chan materially the composition of the urine. This continues to contain i full relative quantity of solid matter.

It is very difficult to find a satisfactory plan of classifying the remai ing forms of chronic Bright's disease. The changes found in all tl kidneys are of much the same kind, although differing in degree. Tl clinical symptoms also are of much the same character in all the patient And yet it has always been felt by clinical observers that there are thr main forms of chronic Bright's disease, and that there must be anat mical differences to correspond to these forms.

It has seemed to me that the simplest form of classifying these ki neys is according to the presence or absence of exudation from the blood vessels, and that, of the non-exudative cases, we may distinguish th more active and the very slow cases. In this way, we may speak o chronic diffuse nephritis with exudation; chronic diffuse nephriti without exudation; and chronic diffuse nephritis of very slow develop ment.

3. *Chronic Diffuse Nephritis with Exudation.*—This follows acut and subacute diffuse nephritis and chronic degeneration of the kidney or it is from the first a chronic lesion.

There is a certain activity about the morbid process, although th patients may not die until after a number of years. The disposition t dropsy, to anæmia, to loss of flesh and strength, is very marked.

The specific gravity of the urine is low, but yet often not below 1020. The excretion of urea is diminished. The quantity of urine is increased, except at the times when the kidney is congested. Albumin in large quantities is present, except at the times when the inflammation seems nearly to have subsided. White and red blood-cells are present from time to time when the nephritis becomes more intense.

Most of the kidneys are increased in size. The surface is smooth or roughened, or nodular. The cortex is thick and white, or gray, or mottled with various combinations of white, yellow, gray, and red. Less frequently the kidneys are small, the cortex is thick, white or gray. Occasionally the size and gross appearance are hardly to be distinguished from those of a normal kidney.

The changes in the kidney are: a large exudation from the blood-vessels, most of which is mixed with the urine, while a part is found in the kidney tubules in the form of coagulated matter and casts; degeneration of the renal epithelium; a growth of new connective tissue in the stroma between the tubes; swelling and multiplication of the tuft cells of the glomeruli; obliteration of the capillaries of the glomeruli, sometimes waxy degeneration of these capillaries. In a considerable number of these kidneys, the walls of the arteries are thickened or the seat of waxy degeneration.

As regards the exact detail of the development of these changes, the variety is almost without limit. The more ordinary types of the lesion are as follows:

(*a*) The kidneys are changed as they are in acute diffuse nephritis. There are the same cortical wedges containing new connective tissue, atrophied tubes and glomeruli, arteries with thickened walls, and glomeruli with a new growth of capsule cells. Between the wedges the tubes are more or less dilated; their epithelium is flattened; there is a diffuse growth of connective tissue in the stroma; the tuft cells of the glomeruli are increased in size and number.

(*b*) There is a diffuse growth of connective tissue in the stroma of the cortex. The tubes are more or less dilated, their epithelium is flattened. The tuft cells of the glomeruli are increased in size and number.

(*c*) There are irregular areas, just beneath the capsule, composed of connective tissue. atrophied tubes, and atrophied glomeruli. In the cortex between these areas there are more or less degeneration of the epithelium, irregular thickening of the stroma, deformities of the tubes, growth of tuft cells, and atrophy of the glomeruli.

(*d*) There is waxy degeneration of the glomerular capillaries and arteries added to the other lesions.

(*e*) In addition to the other lesions there is extravasated blood in the tubes.

4. *Chronic Diffuse Nephritis without Exudation.*—The difference between the more rapid and the very slow forms of chronic nephritis without exudation is a clinical one. The lesions are the same in both sets of cases.

In the more rapid cases, the ordinary symptoms are loss of flesh a strength, dyspnœa, contraction of the arteries, headache, sleeplessne convulsions, and coma. The clinical history extends over a few mont or over many years. The urine is generally abundant and of low spe fic gravity and without albumin. At the times when the patient worse, the urine is apt to be diminished and to contain a little albumi

In the slow cases, there may be no symptoms, except a gradual fall the specific gravity of the urine, up to the time of the patient's death; from time to time some of the symptoms just mentioned are develope

After death many of these kidneys do not differ in their gross a pearance from normal kidneys. If the disease is far advanced, howeve

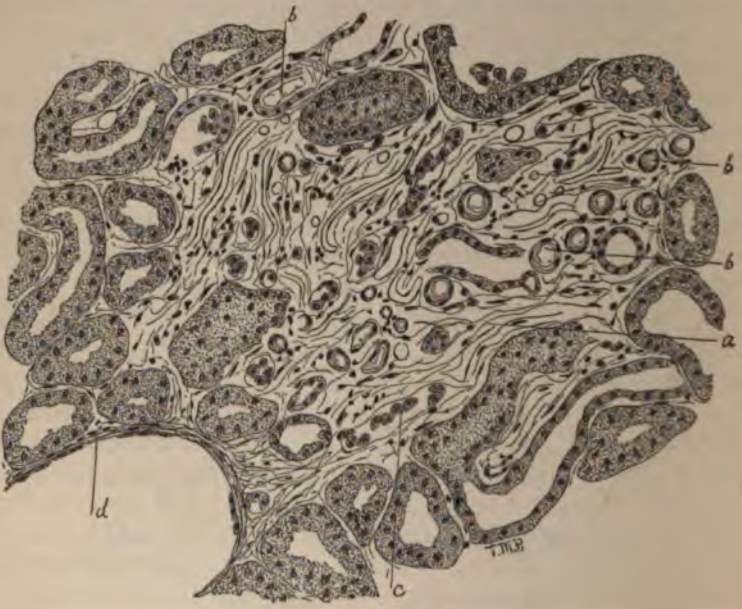

FIG. 182.—CHRONIC DIFFUSE NEPHRITIS.

Atrophied kidney, showing small patch of new connective tissue with atrophy of inclosed tubul *a*, new connective tissue; *b*, atrophied tubule containing hyaline cast; *c*, tubule with epitheliu peeling off; *d*, thickened capsule of glomerulus.

most of them are atrophied, with adherent capsules and nodular surface Less frequently these kidneys are very large.

The most prominent feature of the lesion is the growth of new co nective tissue in the stroma (Fig. 182). This is confined to areas irregular shape in the cortex, or there is also a diffuse growth of co nective tissue in the cortex and in the pyramids.

The epithelium of the tubes is degenerated. Where there is muc new connective tissue the tubes are atrophied and compressed. In oth places the tubes are irregularly dilated, sometimes so much so as form cysts.

Of the glomeruli, some have their capsules thickened, some show a swelling and growth of the tuft cells, some are completely atrophied; in some the capillaries undergo waxy degeneration (Fig. 183).

There are very often changes in the arteries. There may be only thickening of the muscular coat, with no change in the size of the lumen of the artery. There may be a diffuse connective-tissue thickening of the entire wall of the artery, with moderate diminution of its lumen. There may be thickening of the inner coat so great as partly or completely to close the artery.

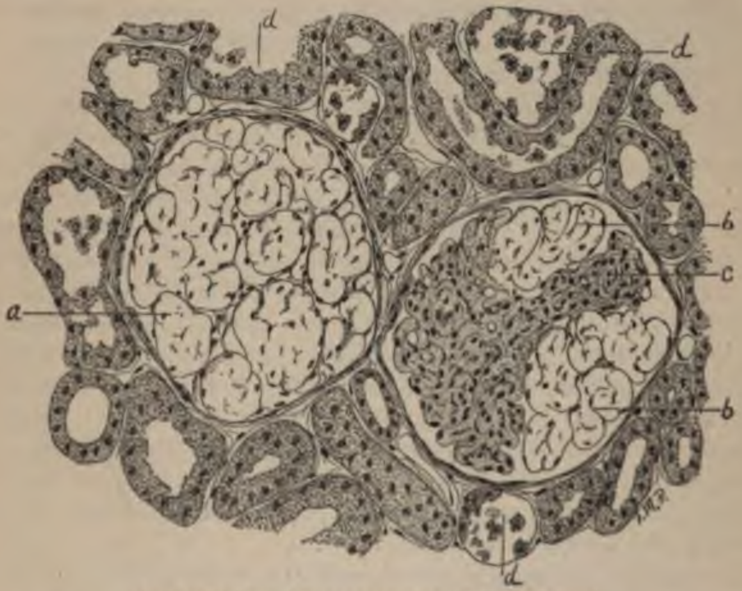

FIG. 183.—WAXY DEGENERATION OF TUFT CAPILLARIES.

a, the tuft is completely transformed into a waxy mass; *b*, portions of tuft waxy; *c*, tuft capillaries normal; *d*, convoluted tubule with disintegrating epithelium.

SUPPURATIVE NEPHRITIS AND PYELO-NEPHRITIS.

Suppurative inflammation of the kidney may be produced by injuries, by emboli, by cystitis, and may occur without discoverable cause.

1. *Suppurative Nephritis from Injury.*—Gunshot wounds, incised or punctured wounds, falls, blows, and kicks are the ordinary traumatic causes. If the injury is a very severe one, it usually causes the death of the patient in a short time; if it is less severe, suppurative inflammation is developed. The inflammatory process may be diffuse, so that nearly the whole of one or of both kidneys is converted into a soft mass composed of pus, blood, and broken-down tissue; or it is circumscribed and one or more abscesses are formed in the kidney.

2. *Embolic Abscesses.*—In pyæmia and in malignant endocarditi small infectious emboli find their way into the arteries of the kidne and produce necrosis of small areas of tissue, with surrounding zones suppurative inflammation. The entire kidney is enlarged and congeste and is dotted with little white foci surrounded by red zones. The fo are formed by an infiltration of pus cells between the tubes, with mo or less degeneration of kidney tissue. Colonies of micrococci are som times, but not always, found in the Malpighian tufts and in the abscess (see Fig. 31, page 93).

3. *Idiopathic Abscesses.*—Sometimes abscesses of one or both kidne are met with which have existed for a long time and for which r cause can be discovered. After death the kidney is found changed int a sac full of pus and surrounded by fibrous tissue. The pelvis an calyces are dilated, and their walls are thickened. The connective ti sue around the kidney, and its capsule, are also thickened. Suppura ing sinuses may extend from the kidney into the surrounding soft part

4. *Suppurative Pyelo-Nephritis with Cystitis.*—Both kidneys ar usually affected. The mucous membrane of the pelvis is congeste thickened, and coated with pus or with patches of fibrin. Scattere through the kidneys are abscesses and foci of pus of different sizes. Th smallest are hardly visible to the naked eye, but with the microscope w find small collections of pus cells between the tubes, with swelling an degeneration of the renal epithelium. The larger purulent foci loo like white streaks or wedges parallel to the tubes and surrounded b zones of congestion. The larger abscesses replace considerable portion of the kidney tissue.

The ureters are sometimes inflamed, their walls are thickened, thei inner surfaces are coated with pus or fibrin.

The bladder is always inflamed, and this is the primary lesion, t which the kidney lesion is secondary. It may present any of the lesion of acute or chronic cystitis.

CHRONIC PYELO-NEPHRITIS.

Chronic cystitis or calculi in the pelvis of the kidney may set up a chronic inflammation which involves both the pelvis and calyces and the kidney tissue. The mucous membrane of the pelvis and calyces is thickened, the epithelial layer is changed, there is a growth of granulation tissue beneath the epithelium, and there may be little polypoid outgrowths. The surface of the mucous membrane is coated with pus or fibrin, or the cavity of the pelvis is dilated and distended with purulent serum.

The kidney itself is the seat of a chronic interstitial inflammation with the production of new connective tissue, and sometimes of pus with obliteration of the renal tubules.

TUBERCULAR NEPHRITIS.

This lesion is usually, though not always, associated with tubercular inflammation in other parts of the genito-urinary tract.

It is usually unilateral, occurring most frequently on the left side. The process may commence in the kidney or in some other part of the genito-urinary tract. If only one kidney is involved, the other is apt to become the seat of chronic diffuse nephritis, with waxy degeneration of the walls of the arteries. The tubercular inflammation may occur in a kidney already the seat of chronic inflammatory changes.

The lesion seems to begin in the mucous membrane of the pelvis and calyces, and extend from thence first to the pyramidal and afterward to the cortical portion of the kidneys. In the mucous membrane of the pelvis and calyces, there is a growth of granulation tissue studded with tubercle granula in the stroma, while the epithelial cells proliferate, become deformed, and desquamate. This process is often rapidly succeeded by cheesy degeneration of all the inflammatory products.

In the kidney, there is the same production of granulation tissue and tubercle granula, which soon undergo cheesy degeneration, the degeneration involving the adjacent kidney tissue. In addition to this, there is in the rest of the kidney chronic interstitial or suppurative inflammation. So the entire kidney is enlarged, portions are in the condition of cheesy degeneration or have sloughed away, while the rest of the kidney is dense and hard. Or, if suppuration takes place, the kidney is hollowed out into cavities filled with cheesy matter and pus.

Sometimes the process comes to a standstill, and then the cheesy portions are infiltrated with the salts of lime.

EMBOLISM AND THROMBOSIS.

Acute and chronic endocarditis affecting the left side of the heart, and chronic endarteritis of the aorta, frequently result in the formation of vegetations, portions of which become detached and lodged as emboli in the branches of the renal artery.

The occlusion of an artery in this way produces in the kidneys wedge-shaped infarctions, varying in their size with the size of the obstructed artery. The infarction loses the natural red color of the kidney, and becomes first yellow and then white. The renal epithelium degenerates and disappears, the tubes become collapsed and shrunken; around the infarction is a zone of congestion and of infiltration with pus cells. After this the infarction becomes shrunken, dense, and changed into connective tissue. The kidney is then left deformed by the cicatricial depressions and contractions. It is possible, however, for the infarction to become gangrenous, or to be surrounded by a zone of purulent infiltration, and break down so as to form an abscess. Rarely the infarctio are of the hæmorrhagic variety.

Embolism of the trunk of the renal artery produces complete necro of the kidney.

Infectious emboli are small, and produce little purulent foci (above).

Thrombosis of the renal vein and its branches may occur in patien suffering from chronic Bright's disease.[1] It can also be produced tumors pressing on the veins, by thrombi of the vena cava; and occu as a primary lesion dependent on the general condition of the patient.

HYDRONEPHROSIS.

Dilatation of the pelvis and calyces of the kidney is found as a co genital condition. In some cases, other malformations, such as club-foo hare-lip, and imperforate anus, are also present. The pelves and calyc of both kidneys and the ureters are distended with urine; the bladder also distended and its wall may be hypertrophied. The urethra may b closed, or no obstruction can be demonstrated. In these latter cases, is supposed that there does exist some membranous obstruction, which broken by the probe or catheter used to explore the urethra.

In adult life, hydronephrosis is produced by mechanical obstructio of the urethra or ureters, due to inflammation, tumors, or calculi. According to the position of the obstruction, either one or both kidneys ar involved.

The pelvis and calyces are dilated, sometimes enormously, and fille with urine alone, or urine mixed with pus. The kidney tissue is flattened and thinned over the distended cavities. Its texture may remain unchanged, or there may be developed suppurative pyelo-nephritis or chronic diffuse nephritis.

THE CYSTIC KIDNEY.

Cysts are formed in the kidneys, both during intra-uterine and extra-uterine life.

The congenital cystic kidney is a very remarkable pathological condition. Either one or both kidneys are enormously enlarged and converted into a mass of cysts. The cysts are of all sizes and are separated from each other by fibrous septa or compressed kidney tissue. They contain a clear, yellow, acid fluid, holding in solution the urinary salts. Or the fluid is turbid and brown, and contains blood, uric acid crystals, and cholestearin. The cysts are lined with a single layer of flat polygonal cells. They seem to be formed by a dilatation of the tubules and of the capsules of the Malpighian bodies. As causes for such dilatations are found obliteration of the tubes in the papillæ, and stenosis of the pelvis, ureters, bladder, or urethra. Other congenital malformations are often associated with this one.[1]

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[1] *Moxon*, Trans. Lond. Path. Society, 1870, p. 248.

In adult life we find three varieties of cystic kidney:

1. In kidneys which are otherwise normal, there are one or more cysts filled with clear or brown serum or colloid matter. These cysts do not appear to interfere at all with the function of the kidneys.

2. In chronic diffuse nephritis, especially in the atrophic form, groups of tubes are dilated. Apparently one or more of the larger tubes in the pyramids is obstructed, and this causes dilatation of a corresponding group of tubes. Such a dilatation may be moderate in size, or it may form cysts visible to the naked eye.

3. Both kidneys are very much enlarged and converted into a mass of cysts containing clear or colored serum or colloid matter. The nature of these cysts is uncertain. It is possible that they are congenital. They are sometimes associated with similar cysts in the liver. They seem to produce no renal symptoms until shortly before the patient's death, unless chronic nephritis also exists, and then there are the ordinary symptoms of chronic Bright's disease.

PERINEPHRITIS.

The loose connective tissue which is situated around and beneath the kidney may become the seat of suppurative inflammation, and in this way abscesses of considerable size are formed.

Such a perinephritis may be either secondary or primary. The secondary cases are due to extension of the inflammation from abscesses in the vicinity, such as are formed with caries of the spine, pelvic cellulitis, puerperal parametritis, perityphlitis, and suppurative nephritis.

The primary cases occur after exposure to cold, after contusions over the lumbar region, and after great muscular exertion; or no cause can be discovered.

Complicating cases occur in the course of typhus and typhoid fevers and of small-pox.

Most of the reported cases have been in persons between the ages of twenty and forty years. Less frequently children and older persons are affected.

In the idiopathic cases, the connective tissue behind the kidney seems to be the point of origin of the inflammatory process, and it is here that the pus first collects. After the abscess has formed, the suppuration extends and the pus burrows in different directions: backward through the muscles; downward into the iliac fossa, the perineum, the bladder, the scrotum, or the vagina; forward into the peritoneal cavity or the co upward through the diaphragm.

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[1] Virch., Ges. Abhandl.

The kidney itself is simply compressed by the abscess, or its ti becomes involved in the suppurative inflammation.

RENAL CALCULI.

In the kidneys of new-born children, from the first to the fourtee day after birth, the large tubes of the pyramids often contain sm brownish, rounded bodies composed of the urates of ammonium a sodium. Similar masses may also be present in the calyces and pelv In still-born children these masses are usually absent. The carbon and phosphate of lime may be deposited in the tubes of the pyramids, the form of white linear masses, in the kidneys of old persons and those who have suffered from destructive diseases of the bones.

Urate of soda in the form of acicular crystals is deposited both in t tubes and stroma of the kidneys of gouty persons.

Concretions of the urinary salts are often formed in the pelves of t kidneys. They may remain there as rounded masses, or they may atta a large size and be moulded into the shape of the pelvis and calyce Smaller calculi may pass into the ureter and either become impact there or pass through it into the bladder. The most common form calculus is that composed of uric acid. But they may also be formed uric acid with a shell of oxalate of lime, or of oxalate of lime alon or of the phosphates, or of cystin.

The most serious result of the presence of these calculi is the occl sion of the ureters or the production of pyelo-nephritis.

TUMORS.

Fibroma.—Small, hard, white fibrous nodules are frequently foun in the pyramids. They are of no special importance. They may b mistaken for miliary tubercles. Large fibromata are very rare.[1]

Lipoma.—Small fatty tumors are found in the cortex of the kidne just beneath the capsule. They are composed of fully developed fa tissue. The fat is developed in the stroma so as to replace the kidne tissue.[2]

Papilloma.—Villous tumors formed of tufts of connective tissue covered with epithelium may grow from the mucous membrane of the pelvis.[3] A peculiar form of papillary and cystic growth of the ureter is described.[4]

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[1] *Wilks*, Trans. Lond. Path. Soc., xx.

[2] *Virchow*, "Krank. Geschwülste," Bd. 1, p. 385.

[3] Trans. Lond. Path. Soc., 1870, p. 239.

[4] Virch. Arch., Bd. 66, p. 139.

Myxo-Sarcoma.—Large tumors may grow from the pelvis of the kidney. They are not simple myxomata, but are composed of mucous tissue, fat, and sarcomatous tissue.

Myoma.—Small tumors composed of smooth muscular fibres and of round cells are found in the cortex close to the capsule.

A tumor composed of striated muscle and round cells is described by Cohnheim.[1]

A tumor composed partly of smooth muscle, partly of striped muscle, and partly of sarcomatous tissue, is described by Eberth.[2]

Angioma cavernosum occurs in the form of small nodules situated in the cortex.

Lymphoma.—Small white tumors composed of tissue like that of the lymphatic glands are found in cases of leukæmia and pseudo-leukæmia. Less frequently they are found with typhoid fever, scarlet fever, and diphtheria.

Adenoma.—This form of tumor is situated in the cortex of the kidney, and may invade the pyramidal portion also. Usually there is only a single tumor, but sometimes two or more, or they may even occur in both kidneys. They vary in size; some are not larger than a pea, others are as large as a hen's egg. They are of rounded form, of whitish color, and separated by a capsule from the kidney tissue. The tumors are most frequent in persons over forty years of age.

There are two principal varieties of these tumors, the papillary and the alveolar.

1. *The Papillary Adenoma.*—There are cavities of different sizes, from the walls of which spring branching tufts covered with cylindrical or cuboidal epithelium. These tufts nearly fill the cavities.

2. *The Alveolar Adenoma.*—There is a connective-tissue framework inclosing small round, oval, or tubular alveoli, lined or filled with cells. The cells are large, polygonal, nucleated bodies.

The adenomata frequently undergo fatty degeneration, which may be so complete that they look like fatty tumors.

Or there may be an excessive development of the stroma, with atrophy of the epithelial cells.

There may be an excessive development and dilatation of the capillaries and veins in the stroma.

Cysts of considerable size may be formed by dilatation of the cavities or alveoli.[3]

There are larger tumors involving the whole of the kidney, and accompanied with metastatic growths in other parts of the body, which have the same structure as the papillary adenomata.

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[1] Virch. Arch., Bd. 65, p. 64.

[2] Virch. Arch., Bd. 55, p. 518.

[3] Medizinisch. Jahrb., 1883, p. 213. Virch. Arch., Bd. 93, p. 39.

A congenital adenoma is described by Weigert.[1]

Carcinoma.—Besides secondary carcinoma of the kidney, t also a primary form. Our knowledge of this has been much ob by confounding with it adenomata and sarcomata.

There seems to be, however, a real epithelial growth, origina the kidney tubules, which forms tumors of large size and mal character.

Sarcoma.—Tumors formed of connective-tissue cells may or either in the pelvis of the kidney or in the kidney itself. The tumors of large size and malignant character. Those which grow the pelvis are usually myxo-sarcomata. Those which originate kidney tissue reach a large size and are soft and hæmorrhagic. stroma forms irregular alveoli filled with small round cells.

PARASITES.

Echinococcus, in its ordinary form of mother and daughter c sometimes found in the kidney. The cysts may open into the p the kidney, into the pleura, or through the wall of the abdomen.

Cysticercus cellulosæ is of very rare occurrence.

Pentastomum denticulatum has been seen once by E. Wagner

Filaria sanguinis hominis is found in the arteries, veins, lymp and stroma.

Strongylus gigas has been found several times in the pelvis kidney.

THE URINARY BLADDER.

MALFORMATIONS.

Extroversion of the bladder is one of the most frequent mal tions, and may occur in either sex. It presents several varieties:

1. The umbilicus is lower down than usual, the pubic bones united at the symphysis, the pelvis is wider and shallower than it be. Between the umbilicus and pubes the abdominal wall is w In its place is a projecting, ovoid mass of mucous membrane, in may be seen the openings of the ureters. The penis is usually ru tary; the urethra is an open fissure (epispadia); the clitoris may b rated into two halves. The ureters usually open normally; son their openings are displaced or are multiple. They may be dilat

2. There may be a fissure in the abdominal wall, filled up perfectly-formed bladder.

3. The umbilicus may be well formed, and there is a portion dominal wall between it and the extrophied bladder.

4. The external genitals and urethra may be well formed, a symphysis pubis united, while only the bladder is fissured.

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[1] Virch. Arch., Bd. 67, p. 492.

5. The genitals, urethra, and symphysis may be well formed, the bladder closed except at the upper part of its anterior wall. The bladder is entirely or in part inverted and pushed through the opening in the abdominal wall.

The *Urachus* normally remains as a very small canal, five to seven cm. long, with a small opening into the bladder, or entirely closed at that point. If there is a congenital obstruction to the flow of urine through the urethra, the urachus may remain open and the urine pass through it.

Absence of the Bladder is of rare occurrence. The bladder may be very small, the urine passing almost directly into the urethra. The bladder may be separated into an upper and a lower portion by a circular constriction. It may be completely divided by a vertical septum into two lateral portions. Diverticula of the wall of the bladder are sometimes found in new-born children. Partial or complete closure of the neck of the bladder may occur. This may lead to hydronephrosis, or the urine may be discharged through the open urachus.

CHANGES IN SIZE AND POSITION.

Dilatation.—This may be *general* or *partial*, leading to the formation of diverticula.

General dilatation of the bladder is produced by the accumulation of urine in consequence of some mechanical obstacle to its escape, or of paralysis of the muscular walls of the organ. The dilatation is usually uniform and may be very great, so that the bladder may reach to the umbilicus. If the walls of the bladder are paralyzed; or the obstruction occurs suddenly or is complete, the wall of the bladder is thinned. When an incomplete obstruction exists for some time, the walls of the bladder are apt to hypertrophy, so that, although the bladder is larger than normal, the walls may not only be of the usual thickness, but even very much thicker. In the fœtus, dilatation of the bladder may reach such a size as to interfere with delivery.

The retained urine in dilated bladders is liable to decomposition, leading to inflammation or gangrene of the mucous membrane.

Diverticula of the bladder may be produced by the pouching-out of circumscribed portions of the wall of the bladder, the wall of the pouch containing all the layers of the bladder wall. More frequently, however, they are produced by a protrusion of the mucous membrane between hypertrophied bundles of muscle fibres. They may be very small, or they may be as large as a child's head. They may communicate with the bladder by a large or a small opening. The decomposition of stagnant urine in diverticula is apt to induce inflammation. Calculi may be formed in them or may slip into them from the bladder.

Hypertrophy of the muscular coat of the bladder is usually produced by mechanical obstructions to the outflow of urine, such as strictu the urethra, enlarged prostate, calculi, new growths, etc. The musc coat is thickened uniformly or assumes a trabeculated appearance. organ retains its normal capacity, or is dilated, or becomes smaller. mucous membrane is frequently the seat of chronic or acute inflam tion. Dilatation of the ureters and hydronephrosis frequently accom this condition.

Herniæ of the bladder sometimes accompany intestinal he through the inguinal and crural canals and the foramen ovale. changes in position of the bladder, produced by displacements of vagina and uterus, will be mentioned with the lesions of those orga

In the female, the base of the bladder may press downward, cau protrusion of the vaginal wall (*vaginal cystocele*); or there may be i sion and prolapse of bladder through the dilated urethra.

RUPTURE—PERFORATION.

Penetrating wounds of the bladder may permit escape of urine the abdominal cavity, or infiltration into the surrounding conne tissue, or permanent fistulæ. Such wounds are always serious and quently fatal, owing chiefly to the severe and often gangrenous inf mation which decomposing urine sets up in the connective tissue, the peritonitis induced by the same cause.

Rupture of the bladder may be produced by severe blows and when the bladder contains urine. More rarely, rupture takes place over-distention. Death may occur from rupture of the bladder escape of urine into the peritoneal cavity, without evidences of perito

Perforations of the bladder are produced by ulceration and gang by abscesses from without, and by cancerous ulceration from the ad ing organs. Fractures of the pelvic bones may produce laceration o bladder. Perforations of the bladder may lead to the establishme fistulæ, communicating with the rectum, vagina, uterus, or ope externally.

DISTURBANCES OF CIRCULATION.

Hyperæmia.—Aside from active hyperæmia of the mucous mem in acute inflammation, the bladder is not infrequently the seat of ch congestion from obstruction to the venous circulation. Under conditions, there may be chronic catarrhal inflammation, or a ma dilatation of the veins (vesical hæmorrhoids), which may give ri hæmorrhage, or to obstruction of the opening of the ureters.

Hæmorrhage.—Extensive hæmorrhages into the bladder are monly due to injury or to the presence of calculi or tumors. hæmorrhages into the substance of the mucous membrane may ac pany inflammation, the hæmorrhagic diathesis, scurvy, purpura, s pox, etc. If the hæmorrhage is considerable and occurs rapidly in an empty bladder, a clot is apt to form; but when the blood mixes with urine as it is extravasated, it more commonly remains liquid and is discharged as a reddish-brown fluid.

INFLAMMATION.

Acute Catarrhal Cystitis.—This may be incited by the presence of urine which has decomposed under the influence of bacteria; by cantharides or other drugs; by the presence of foreign bodies and calculi; or it may be due to an extension of gonorrheal urethritis or vaginitis; or it may occur without assignable cause. The mucous membrane is swollen and congested, although these alterations may not be very evident after death. The surfaces may be coated with mucus containing red blood-cells and pus. The epithelium is apt to be loosened and in some places peeled off, so that superficial or deep ulceration may occur. We may find mixed with the urine in the organ shreds of mucus, pus cells, epithelial cells of various shapes, usually more or less swollen and granular, or fragments of such cells; red blood-cells and bacteria. Resolution may occur from acute catarrhal cystitis, but it very frequently assumes a chronic character.

Chronic Cystitis.—In this form the mucous membrane may be swollen, succulent, grayish, or mottled with spots of congestion or extravasation, and covered with a layer of mucus and pus. Microscopically the membrane may be more or less infiltrated with pus cells, and pus may be constantly produced and thrown off into the urine. Later the mucous membrane may become thickened either diffusely or in the form of tufts or polypi. In some cases it becomes atrophied. Owing to decomposition of the hæmoglobin in the extravasated blood, the mucosa may become pigmented, brown, or slate-colored. The mucous membrane frequently becomes eroded, especially on the most elevated portions, or deep ulcerations may occur. The muscular coats may become paralyzed and the bladder dilated; or the submucosa or the muscularis, or both, may become hypertrophied. The mucous membrane may become incrusted with urinary salts.

In another class of cases, the inflammation assumes a more intense and necrotic character. Larger and smaller shreds and patches of the mucosa die, become brown or gray in color, loosen or peel off, and become mixed with the urine and exudations. The gangrenous process may extend to all the coats of the bladder, so that perforation and fatal peritonitis may occur. The gangrenous form of cystitis is most apt to occur in paralytics. In still another class of cases, the inflammation assumes a suppurative character. The submucosa, the intermuscular connective tissue, and the adjacent parts become infiltrated with pus, either diffusely or in the form of larger and smaller abscesses, which

This form of inflammation may occur in connection with s fectious diseases—measles, diphtheria, scarlatina, typhoid fever nection with similar inflammation of the external genitals, in p fever, noma, and sometimes in the presence of foreign bodie rarely an idiopathic disease.

Tubercular Inflammation.—This disease commences by the f of miliary tubercles in the mucous membrane of the bladder. coalescence of the tubercles and the degeneration of tissue abo ulcers are formed, and it is most frequently in the ulcerative s the lesion is seen. The ulcers, which may be large or small, ar most abundant at the base of the organ. Their edges may b and miliary tubercles in greater or smaller numbers are usually the mucosa about them. Not infrequently large shreds of t loosened and cast off. The mucosa about the ulcers is apt to trated with small spheroidal cells. Tubercle bacilli are present of the tubercles, and in the edges and base of the ulcers. T also be found in the urine, and are then of diagnostic sig Catarrhal inflammation is a very constant accompaniment of th Tubercular cystitis may occur in connection with tubercular i tion of the lungs, intestines, or of the kidney, uterus, prostate,

TUMORS.

Fibromata have been described, occurring as small nodular t the submucosa, but they are rare.

Aside from the polypoid thickenings of the mucosa occu chronic cystitis, soft vascular *papillomata* are of frequent oc and is then rarely due to metastasis, but usually to an extension of the growth from neighboring parts, as the uterus, vagina, or rectum.

Primary carcinoma of the bladder may occur:

1. As a diffuse *scirrhous infiltration* of the entire wall of the bladder, usually with ulcerations of its inner surface.
2. As a *circumscribed nodule* which grows inward and outward, ulcerating on its inner surface, and sometimes producing perforations.
3. As *villous* or *papillomatous growth*. The tumor grows from one or more points of the inner surface of the bladder. It is formed of tubular follicles lined with cylindrical epithelium, and, on its inner free surface, of tufts covered with cylindrical epithelium. The new growth may involve the entire thickness of the wall of the bladder.
4. A few cases of carcinoma have been described in which the stroma contained a varying quantity of smooth muscle tissue.[1]

Cysts.—Dermoid cysts of the wall of the bladder have been described, but are rare. Small cysts with serous contents sometimes occur in the mucous membrane. A part of them, at least, are believed to be due to faulty embryonal development.

PARASITES, ETC.

Among the animal parasites occasionally found in the bladder may be mentioned *Echinococcus, Distoma hæmatobium, Filaria sanguinis, Ascarides*, and *Oxyurides*.

Bacteria of various forms not infrequently occur in the bladder, particularly in connection with chronic cystitis. *B. termo, Micrococcus ureæ*, and *Sarcina* may be mentioned as of most frequent occurrence.

A great variety of foreign bodies may be found in the bladder, particularly in the female. If their stay is long, they are apt to become incrusted with urinary salts.

CALCULI.

Vesical calculi may occur singly or in great numbers, and vary greatly in size, ranging from small, sand-like particles up to masses four or five inches in diameter, but the usual range is from the size of a pea to that of a hen's egg. They are usually oval, spheroidal, or elongated; or, when several are present, they are apt to be faceted. The surface may be smooth or rough. They are usually more or less distinctly lamellated, and are frequently formed around a central body called a nucleus, which may either be formed of urinary salts or some foreign body. Their most common constituents are *phosphates, uric acid* and *urates*, and *calcium oxalate*, or various combinations of these.

Uric Acid Calculi.—These are the most common of vesical calculi.

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[1] The literature of tumors of the bladder may be found in *Stein's* "Study of the Tumors of the Bladder," 1881.

In the form of small brownish-red, crystalline aggregations they may passed as "gravel." The larger uric acid calculi are not commonly very great size, are frequently finely nodulated on the surface, but m be smooth. The color varies from light yellow to dark reddish-brow they are usually dense and lamellated.

Calculi formed of Urates.—Calculi composed of pure urates are ra these salts being more commonly combined with uric acid and the ph phates to form the complex calculi. Sodium urate, in the form of sm spined, more or less globular crystalline masses, forms one of the var ties of "gravel."

Phosphatic Calculi.—Pure *calcium phosphate* calculi are rarely foun as whitish, usually smooth, and small lamellated concretions.

Mixed or Triple Phosphate calculi are common, and frequently atta large size. These calculi are sometimes pure, but the deposit is mo frequently associated with other salts, either as incrusting or intercalat lamellæ. Triple phosphate calculi are usually rough on the surface, grayish-white color, lamellated, and frequently very friable.

Small gray or white, hard, and usually smooth calculi of pure *calciu carbonate* occur rarely. Calcium carbonate is sometimes passed as grav in the form of minute spheroidal bodies, either singly or in clusters.

Calcium Oxalate calculi (mulberry calculi) are comparatively con mon, either pure or in combination with uric acid or the phosphate Calcium oxalate may occur in the form of very small, hard, smooth cor cretions, or as larger, heavy, hard, finely or coarsely nodulated brown o blackish lamellated masses. The nucleus or some of the lamellæ, o both, are often composed of uric acid.

Cystin Calculi are usually ovoidal in shape, of waxy consistence, o clear or brownish or greenish-yellow color, with mammillated surface an crystalline fracture. Cystin may be associated in a variety of ways wit other calculi.

Xanthin Calculi, which are very rare, are usually of moderate size smooth, of a cinnamon or cinnabar-red color, lamellated, and oval o flattened in shape.

Solid masses of fibrin and blood sometimes occur in the bladder, and may exist as independent structures, or form nuclei for the deposit of urinary salts.

For a detailed account of calculi, the conditions under which they form, modes of analysis, etc., we refer to special works on this subject.

THE URETHRA.

CONGENITAL MALFORMATIONS.

Some of the malformations of the urethra are described with those of the penis.

The urethra may be impervious, or may open at the root of the penis. More commonly there is partial obliteration or stricture of some part of the canal.

The entire urethra may be dilated into a sac full of urine.

There may be a canal on the dorsum of the penis, formed by the fusion of the spermatic cords, and opening in the glans above the urethra.

There may be two or more openings of the urethra.

The canal may be dislocated so as to open in the inguinal region.

A number of cases have been reported in which a valve in the urethra has produced hypertrophy of the bladder, dilatation of the ureters, and hydronephrosis.[1]

Owing to its narrowness, greater length, and peculiar connections with the internal generative organs, the male urethra is much more liable to disease than the female.

CHANGES IN SIZE AND POSITION.

Dilatation of the urethra may be produced by strictures, or by calculi or other bodies fixed in its lumen. The dilatations are fusiform or sacculated in shape, and may reach the size of an orange or be even larger.

Strictures of the urethra are usually produced by inflammation of its walls.

The stricture may be *temporary*, produced by a diffuse inflammatory swelling of the mucous membrane, or by the raising of the relaxed membrane into a fold or pocket.

Permanent strictures are produced by structural changes in the walls of the urethra.

1. The mucous membrane and submucous tissue are left hard and unyielding by the preceding inflammation. Subsequently the new fibrous tissue contracts and narrows the canal.
2. Ulceration of the mucous membrane leaves cicatricial tissue, which contracts, and also produces adhesions and bands of fibrous tissue.
3. There is fibrous induration of the corpus spongiosum, and consequent constriction of the urethra.

The most frequent position of strictures is at the junction of the membranous and spongy portions of the urethra, or close to this point.

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[1] Virch. Arch., Bd. 49, p. 348.

They also occur at the fossa navicularis and the meatus, but frequen in the prostatic portion. There may be one stricture or several. T consequences of stricture are dilatation of the urethra, the bladder, t ureters, and hydronephrosis; inflammation and ulceration of the ureth behind the stricture, with perforation, infiltration of urine, or the f mation of fistulæ.[1]

The urethra may also be obstructed by folds of the mucous membra by muscular valves at the neck of the bladder; by wounds; by pol and swollen glands; by new growths; by changes in the prostate a perineum; by calculi, mucus, blood, and echinococci coming from t bladder; by foreign bodies introduced from without.

Prolapse and inversion of the mucous membrane is seen in you girls and women in rare cases. There is a bluish-red swelling, from t size of a pea to that of a walnut, at the meatus. In the male, invagin tion of the mucous membrane of the urethra has been seen after injuri of the perineum.

WOUNDS—RUPTURE—PERFORATION.

Wounds of the urethra are produced in many ways, but most com monly by catheters and bougies. The wounds may cicatrize, or the may be infiltration of urine or the formation of fistulæ or false passages

Ruptures of the urethra are produced by severe contusions and b fracture of the pelvic bones. Extravasations of blood and urine, an gangrenous inflammation of the surrounding soft parts, are the ordinar results.

Ulceration and perforation of the urethra may lead to the formatio of fistulæ, which open in various directions through the skin.

INFLAMMATION.

Catarrhal Urethritis may be simple and due to the action of chemical irritants, to the extension to the urethra of inflammation from other parts, and to unknown causes; but it is most frequently due to the action of the gonorrhœal poison. In its acute form it involves either a portion or the whole of the urethra. The mucous membrane is red, swollen, and covered with muco-pus. The inflammation may extend to the fibrous wall of the urethra, the corpora spongiosa and cavernosa. This may result in the formation of new connective tissue or of abscesses, especially near the fossa navicularis. The inflammation may also extend to the bladder, the glands of Cowper, the prostate, the spermatic cord, and the testicles. The inguinal glands also may be swollen and inflamed, and the lymphatic vessels on the dorsum of the penis may be involved in the same process.

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[1] For literature of stricture of urethra and plates illustrating several forms, see article by *Dittel* in Pitha and Billroth's "Handbuch der allg. Chirurgie," Bd. 3, Abth. 3.

Chronic inflammation of the urethra may exist for a long time with the production of a muco-purulent exudation, but without the occurrence of marked structural lesions. In other cases it leads to ulceration, to fibrous induration of the wall of the canal, to induration and swelling of the mucous follicles, to polypoid thickenings of the mucous membrane.

The exudation in gonorrhœal inflammation of the mucous membranes, not only of the urethra but also of the vagina and of the eye, constantly contains, in greater or less numbers, a form of micrococcus which is said by some observers—although this is denied by others—to present characteristic morphological characters.

The micrococcus—called gonococcus—which is spheroidal or ovoidal in shape, usually occurs in pairs or in groups of four or more, and may be contained in the pus cells (Fig. 184) or lie on their surfaces or free in the fluid. The pus cells sometimes contain very large numbers of the micrococci. While the very constant presence of these micrococci justifies the conjecture that they may stand in an etiological relation to the gonorrhœal inflammation, the experiments thus far recorded upon their purification by culture and inoculation have not led to such definite results as to justify us in assigning to them a definite rôle in the disease. The conclusions of investigators are in many respects at variance; the results of animal inoculations are usually negative, and the inoculations thus far practised on the human subject have not been sufficiently extensive and exact.

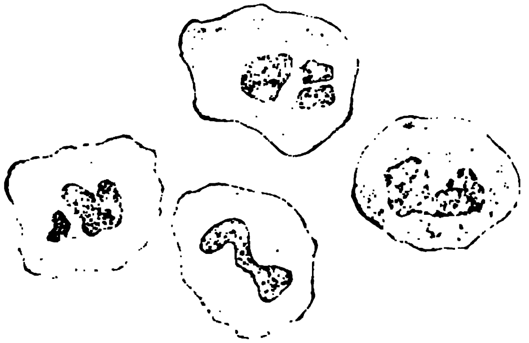

FIG. 184.—GONOCOCCI CONTAINED IN PUS CELLS.
From a case of gonorrhœal urethritis.

The gonococcus may be stained by drying the exudation on a cover glass and using fuchsin or methylin blue.

Croupous Inflammation is sometimes seen in children. Fibrin casts of a small or large portion of the canal may be formed.

Syphilitic Ulcers may be situated at the meatus or as far back as t fossa navicularis. They are apt to produce strictures.

Tubercular Inflammation rarely occurs in the mucous membrane the urethra in connection with tubercular inflammation of the bladd prostate, or testicles.

TUMORS.

Aside from the polypoid outgrowths from the mucous membrane the urethra as the result of chronic inflammation, fibrous polyps m occur congenitally, or polyps containing glandular structures or cy rarely occur. *Carcinoma* may occur as a result of local extension fro adjacent organs or metastasis from the bladder. *Cysts* may occur in t mucous membrane as a result of the dilatation of the mucous gland Circumscribed masses of dilated veins occasionally occur in the urethr forming the so-called *urethral hæmorrhoids.*

The sinus pocularis may be dilated in children by the retention of i secretion, so as to form a tumor which may obstruct the exit of urin cause hypertrophy of the bladder and dilatation of the ureters.

THE ORGANS OF GENERATION.

FEMALE.

THE VULVA.

MALFORMATIONS.

The external genitals may be entirely absent or imperfectly developed. The fissure between the labia may be unformed, or the labia may grow together, with or without obstruction of the urethra. The clitoris and nymphæ may be abnormally large or the nymphæ may be increased in number. The clitoris may be abnormally long, resembling a penis; at the same time the vagina is narrow, the uterus small and undeveloped or malformed; the ovaries small, sometimes situated in the labia; the mammæ small, and the body of a masculine character. Such cases are sometimes called pseudo-hermaphrodites. The clitoris may be perforated by the urethra, or may be cleft and apparently double.

The hymen frequently exhibits various anomalies. It may be entirely absent. The opening may be very large or in unusual places; there may be several openings; the free edge may be beset with papillary projections; there may be no opening at all.[1]

HÆMORRHAGE, HYPERÆMIA, ETC.

Hæmorrhage may take place from wounds or ulcers of the vulva, but the most important form of hæmorrhage is that which occurs in the connective tissue of the labia majora This is produced during labor or from external injury. One of the labia may be swollen and distended by the extravasated blood until it is as large as a child's head. The blood may be gradually absorbed, or it may decompose with suppuration or gangrene of the surrounding tissue. The purulent matter may escape through the skin and the patient recover, or the suppuration may extend into the pelvis and cause death.

A varicose condition of the veins of the labia is not infrequent.

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[1] For description and illustrations of anomalies of the hymen, which may be useful for medico-legal purposes, see *Courty's* "Diseases of Uterus, Ovaries, Fallopian Tubes," Trans. by McLaren, 1883, p. 90.

Œdema may occur in acute form in pregnant and puerperal women. a may terminate in suppuration or gangrene. Œdema of the labia maj frequently accompanies disturbances of the venous circulation, as in c tain heart and lung diseases; or it may occur in chronic diffuse nephri or other wasting diseases, or as a result of thrombosis or other dist bances of circulation in the uterine or perivaginal venous plexus This may be excessive, leading to the transudation of fluid through t skin, to the formation of vesicles, to superficial erosion, or even to ga grene.

INFLAMMATION.

The skin, mucous membrane, connective tissue, and glands of t vulva may be the seat of inflammation. Acute catarrh of the muco membrane may be caused by a variety of irritating influences, but is mo frequently due to gonorrhœal infection. The mucous membrane swollen and red and covered with a muco-purulent exudation. Th labia may be swollen, and the glands are liable to be involved, and a scesses of the labia may be developed. Chronic catarrhal inflammatio may lead to superficial or deep ulceration of the mucous membrane, or t papillary outgrowths, or to thickening of the labia. Suppurative in flammation of the tissue of the labia may occur in connection with similar process in neighboring parts. Erysipelatous inflammation of th skin of the vulva is frequent in young children and may cause death In adults it is less common. Inflammation of the vulvo-vaginal gland may be acute and produce abscesses, or chronic and produce induratio of the gland.

Gangrene may follow erysipelatous inflammation, may occur afte parturition, may accompany severe exhausting and infectious diseases, o may occur as an epidemic disease, especially among children. It may b the result of bruises or other injuries. In some forms, such as thos known as *noma* and *hospital gangrene,* the destruction of tissue proceeds with extreme rapidity.

Herpes, eczema, lichen, prurigo, etc., may be found on the skin of the vulva.

Syphilitic inflammation and ulceration are of frequent occurrence on the vulva, particularly on the mucous surfaces, and frequently lead to considerable destruction of tissue and cicatricial contractions.

Croupous Inflammation may occur with or without diphtheria and a similar lesion of the fauces, and is frequently associated with gangrene.

Lupus.—This form of inflammation, usually with more or less destructive ulceration, occasionally occurs in the vulva.

TUMORS.

Fibroma.—Circumscribed fibrous tumors are found in the connective tissue of the labia, mons veneris, perineum, clitoris, and entrance to the vagina. They may attain a large size, and, attached only by a pedicle, may hang far down between the legs. The skin is usually movable over the surface of these tumors.

Fibroma diffusum (elephantiasis).—This usually involves the clitoris or the labia, or both, and may extend to surrounding parts of the skin. It consists essentially of a diffuse hypertrophy of the skin and subcutaneous tissue, with or without involvement of the papillæ and epidermis. The surface may be smooth or rough. Sometimes when the new growth is circumscribed, rough or smooth polypoid growths, often of large size, are formed. When the papillæ and epidermis are much involved, larger and smaller cauliflower-like excrescences may cover the hypertrophied parts, and the surface be very rough and scaly.

Papillomata.—These growths consist of hypertrophied papillæ covered with thick layers of epithelium. They vary in size from that of a pea to that of an apple, and have a cauliflower appearance.

Syphilitic Condylomata.—In one form, the so-called *mucous patch*, there is an infiltration of the papillary layers of the skin or mucous membrane with variously shaped cells and fluid, so that the tissue has a gelatinous appearance. In other cases, there is an hypertrophy of the papillæ, so that larger and smaller wart-like excrescences are formed. This is called the *pointed condyloma*. *Lipomata*, *fibro-myomata*, and *fibro-sarcomata* are of occasional occurrence in the vulva. A few cases of *melano-sarcoma* are recorded. *Chondroma* of the clitoris has been described. *Carcinoma* of the vulva may be primary, usually in the form of epithelioma of the clitoris or labia, or it may be secondary to cancer of the uterus, vagina, etc.

Cysts are found in the connective tissue of the labia majora and minora. They are from the size of a pea to that of a child's head. They may contain serum, colloid material, purulent or bloody fluid, or they may have the characters of dermoid cysts or atheroma cysts. Their origin is in many cases obscure. In some cases they are doubtless due to dilatation of lymph vessels. Cysts may be formed by a stoppage and filling with fluid of the canal of Nuck, or by a dilatation of the ducts or acini of the vulvo-vaginal glands.

THE VAGINA.

MALFORMATIONS.

The vagina may be entirely absent, and the internal organs of generation also absent or imperfectly developed.

Either the upper or the lower portion of the canal may be absent while the remaining portion is present.

The vagina may be closed by an imperforate hymen or by fibrous septa at any part of its canal. The canal may be abnormally small without being occluded.

The vagina may be double, in connection with a double uterus; or, while the uterus is normal, the vagina may be incompletely divided by a longitudinal septum.

CHANGES IN SIZE AND POSITION.

Dilatation of the vagina is produced by tumors, by the prolapsed uterus, and by the accumulation of blood and mucus behind constrictions or obliterations of the canal. *Lengthening* of the vagina is produced by any cause which draws the uterus upward. *Narrowing* of the vagina is found as a senile change; is produced by tumors and by ulceration of the wall of the canal. Extensive ulcers may even cause entire obliteration of the canal.

Prolapse of the vagina occurs by itself, usually as a result of thickening or laxity of its walls, or in connection with prolapse of the uterus. As an idiopathic process, it usually takes place soon after parturition. A larger or smaller portion of the canal is inverted and projects through the vulva. The entire circumference of the canal may be inverted and prolapsed, or only the anterior or posterior wall. The prolapse is at first small, but may afterwards gradually increase in size, and may drag down the uterus with it. In other cases, prolapse of the uterus is the primary lesion, and the vagina is inverted by the descent of that organ; or the body of the uterus may retain its normal position, while an hypertrophy and lengthening of the cervix alone drag down the vagina.

Hernia vesico-vaginalis—cystocele—may be either the cause or effect of a prolapse of the vagina and uterus. If the cystocele is the primary lesion, it begins as a small projection of the wall of the bladder into the anterior part of the vagina. As the urine accumulates in this sac, it increases in size, projects through the vulva, draws down the vagina and the anterior lip of the cervix, and finally the entire uterus. If the cystocele is the secondary lesion, it is simply produced by the dragging-down of the posterior wall of the bladder by the inverted vagina.

Hernia intestino-vaginalis.—A portion of the intestines may become fixed in Douglas' cul-de-sac between the rectum and uterus. This portion of intestine gradually becomes larger, pushes forward the posterior wall of the vagina, inverts and fills up that canal, and finally projects through the vulva. It may drag with it the posterior wall of the vagina and the uterus.

Rectocele vaginalis.—A sac is formed by the projection of the anterior wall of the rectum and the posterior wall of the vagina. This lesion is of rare occurrence, and does not reach a large size.

When the vagina is prolapsed, there is usually an inflammatory condition of the lining membrane or a thickening of the epidermis.

WOUNDS—PERFORATIONS.

Wounds of the vagina are made by penetrating instruments, by forceps and other obstetrical weapons, and by the fœtus during delivery. Such wounds may heal, may give rise to large hæmorrhages, may suppurate, may produce abscesses in the surrounding tissues, may leave fistulous openings into the vagina, or may cause constriction or obliteration of its canal.

Vesico-vaginal Fistulæ are usually produced by injuries from instruments or from the fœtus during delivery; less frequently by ulceration of the vagina, bladder, or adjacent connective tissue, or by abscess in the surrounding parts. The fistulæ form an opening between either the bladder or the urethra and the vagina. They allow the urine to pass into the vagina. Spontaneous cure does not take place.

Recto-vaginal Fistulæ are formed in the same way as the last-mentioned. They allow the passage of gas or fæces into the vagina. They sometimes heal spontaneously.

INFLAMMATION.

Catarrhal Inflammation of the vaginal mucous membrane may be acute or chronic. It is most frequently caused by gonorrhœal infection, but may be due to local irritation or depend upon general causes. It not infrequently occurs in the new-born. In the acute form, the mucous membrane is swollen and frequently covered with a muco-purulent or a purulent exudation. In the chronic form, the mucous membrane may be swollen, covered with a purulent exudation; there may be an exfoliation of epithelium, shallow or deep erosions, or ulcers. In other cases, the mucous membrane is thickened, dense, and sometimes pigmented, or it may be roughened, covered with papillæ, or it may be relaxed and prolapsed.

Croupous Inflammation may occur after parturition, in dysentery, in typhus and typhoid fever, diphtheria, scarlatina, measles, and other infectious diseases. The mucous membrane is swollen and covered with a grayish layer of fibrin and pus. The mucosa and submucosa may be infiltrated with fibrin and pus. The infiltrated portions of the mucosa and submucosa may die and become gangrenous, and thus deep and extensive ulcers be formed.

Suppurative Inflammation of the fibro-muscular coat of the vagina may occur after injuries or in pregnant and puerperal women. Abscesses may be formed which penetrate into the labia or into the pelvic connective tissue. In other cases, the intense phlegmonous inflammation may lead to the death and casting-off of portions of the vaginal wall, or e of the entire wall.

Gangrene of the vagina may occur as a result of croupous or inte suppurative or syphilitic inflammation, or from unknown causes. the form of *noma* it may be very extensive and rapidly destructive.

Tubercular and *Syphilitic Inflammation*, usually leading to more less extensive ulceration, may occur in any part of the vagina. Tub cular inflammation is secondary to tuberculosis of other parts. Syphili ulcers may heal, sometimes leaving marked cicatrices, and sometimes n

TUMORS.

Fibroma, fibro-myoma, sarcoma, myoma lævicellulare, are of occ sional occurrence in the vagina. *Myoma striocellulare* is of rare occu rence.

Papillomata are of frequent occurrence as a result of chronic inflan mation. *Carcinoma* of the vagina is usually secondary to cancer of th uterus. It may be primary as a circumscribed nodular tumor, or mor frequently it occurs in a papillary and ulcerating form and often spread to neighboring parts.

Cysts.—These are not very common and may be small or as large a a hen's egg. They may be lined with flattened epithelium, and contai serous or viscid, dark-colored or transparent fluid.

PARASITES.

Among the animal parasites, *Oxyuris* and *Trichomonas vaginalis* are of occasional occurrence. Among the vegetable forms *Oidium albicans, Leptothrix* are occasionally seen, while *micrococci* and various other forms of bacteria are common. The pathogenic significance of the bacteria in the vagina is not yet established.

THE UTERUS.

MALFORMATIONS.

The uterus, up to the third month of intra-uterine life, consists of two large cornua, which by the fusion of their lower ends form the uterus.

The uterus, tubes, and vagina may be entirely absent, with or without absence of the external genitals. Or the uterus alone, or the upper part of the vagina also, may be absent.

The uterus may be only rudimentary, while the vagina is normal. It then appears as a flattened solid body with solid cornua. Or there are two cornua joined at their lower extremities so as to form a small double uterus. Or the uterus is represented by a small sac, which may or may not communicate with the vagina. Or there is a very small uterus, with thin muscular walls and two large cornua.

Only one of the cornua which should form the uterus may be developed, while the other is arrested in its growth. The uterus is then a long, cylindrical body, terminating above in one tube. On the side where the other horn should have been developed there is no tube, or only a rudimentary one. Both ovaries are usually present.

The two cornua may be fully developed, but their lower ends remain separated and form a double uterus. An entire separation into two distinct uteri and vaginæ is very rare. More frequently the uterus consists of one body, divided by a septum into two cavities. There are then two cervical portions of the uterus projecting into a single vagina, or each into a separate vagina. Or there is only a single cervix. The septum in the uterus may be complete or only partial.

We also find abnormal size of the uterus, abnormal flexions; the cervix may be solid, or may be closed by the vaginal mucous membrane. Or the cervix may have an abnormal form with a small opening or canal.[1]

CHANGES IN SIZE.

In the new-born infant, the uterus is small, the body flattened, the cervix disproportionately large. During childhood, the organ increases in size, but the body remains small in proportion to the cervix. At puberty the shape changes and the body becomes larger.

At every menstruation the uterus is somewhat swollen and congested. After pregnancy it does not return to its virgin size, but remains somewhat larger. In old age, it gradually becomes smaller; its walls are harder and more fibrous.

Abnormal Smallness of the uterus is sometimes found as an arrest of development. The uterus in adult life retains the size and shape of that of the infant. It may result, however, from chronic endometritis, from repeated pregnancies, from old age, or from chronic exhausting diseases. Its cavity may be smaller than normal, or distended with mucus. Large myomata sometimes cause marked atrophy of the uterine wall. Atrophy of the vaginal portion of the uterus is sometimes observed after repeated pregnancies, sometimes without known cause. Narrowing and obliteration of the cavity of the uterus and of the cervix are usually produced by chronic inflammation.

Enlargement of the Uterus may be due to too early development. It is accompanied by abnormally early development of all the sexual organs and functions. The uterus may be enlarged in connection with heart disease, prolapse and abnormal flexions and versions, chronic inflammations, repeated pregnancies, myomata, and accumulations of bl mucus in the uterine cavity. Enlargement of the vaginal portio be produced by the above causes, and is also found without known One or both lips of the cervix may be uniformly increased in s they may be lobulated.

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[1] Illustrations of various forms of malformation of the cervix may be found in the translation by McLaren of *Courty's* "Diseases of the Uterus, Ovaries," etc., 1883.

Dilatation of the uterus is produced by accumulations of mucus, or pus in consequence of narrowing or obliteration of the or vagina. The uterine walls may retain their normal thickne thickened or thinned. The most frequent position of the stenosis os internum. The retained contents after a time change in char forming a thin. serous fluid—*hydrometra*—or they may be mixe blood. The dilated uterus is not usually larger than an apple, sometimes reaches enormous dimensions. If both os internum a externum are closed, the cervical cavity may be also dilated an uterus have an hour-glass shape. If the obstruction is in the va the uterus and vagina may form a large, flask-shaped body, and th of demarcation between cervix and vagina be lost. In some case dilatation is confined to the cervix. If the obstruction is not com the retained fluid may escape into the vagina and afterward accum again.

Accumulation of menstrual blood in the cavity of the uterus—*h* *tometra*—is usually produced by congenital stenosis of the cervi vagina. The dilated uterus may reach an enormous size. If the is not evacuated by surgical interference. there may be either ruptu ulcerative perforation of the uterus. The blood may escape into abdominal cavity, or be shut in by adhesions. or perforate into the der or intestines. Sometimes the blood passes into the Fallopian tu dilates them, and escapes through their abdominal ends.

CHANGES IN POSITION.

The body of the uterus may become fixed in an abnormal posit while the situation of the cervix is unchanged. The body may be forward—*anteflexion;* backward—*retroflexion;* or sideways—*lat* *flexion.* The flexion may be slight, or so great that the neck and b form an acute angle. Anteflexion is the most common variety, and in which the flexion is greatest. Peritoneal adhesions, flaccidity of uterine walls, particularly after delivery, atrophy of the walls, ova and other tumors, etc., are the usual causes of flexions.

The Versions of the uterus consist in an abnormal inclination of long axis of the organ to that of the vagina. The uterus may be incli backward, forward, or to one side.

Retroversion is very much the most common. The fundus uter directed backward and downward; the cervix, forward and upward. condition is found in various degrees; in the highest, the fundus lie

Douglas' cul-de-sac with the cervix upward, so that the axis of the uterus is parallel to that of the vagina, but in a direction nearly opposite to the normal one. Abnormal looseness of the uterine ligaments, abnormally large capacity of the pelvis, hypertrophy or tumors of the uterus, and pregnancy during the first four months, are some of the more common conditions under which this lesion occurs.

Anteversion.—Inclination of the fundus forward and downward, and of the cervix backward and upward, is not common and seldom reaches a high degree. It occurs under the same general external conditions as anteflexion.

Lateroversion is not very common as a simple lesion, but is not infrequently combined with other displacements. It may be produced by congenital shortening of one of the broad ligaments, by adhesions, or by the pressure of tumors.

The greater degrees of version may produce very grave lesions. The urethra and rectum may be compressed. Cystitis, perforation of the bladder, dilatation of the ureters and hydronephrosis, and fatal obstruction of the bowels may follow. If pregnancy exists, abortion may take place, or the inverted uterus may be forced through the peritoneum and posterior wall of the vagina and project through the vulva. In the non-pregnant uterus, pressure on the veins and consequent chronic inflammation of the organ may follow.

Prolapsus Uteri consists of a descent of the uterus into the vagina. The uterus may be only slightly lowered or it may project at the vulva. In complete prolapse we find a tumor projecting through the vulva, partly covered by the distended vagina, and presenting the opening of the os externum near its centre. The bladder and rectum may be drawn down with the vagina or may remain in place. The exposed cervix and vagina usually become inflamed and sometimes ulcerated, or the mucous membrane may become thickened. The lesion is frequently complicated by hypertrophy of the cervix.

Gradual prolapse, which is most frequent, may be due to an increased weight of the uterus, as in pregnancy, inflammatory enlargement, the presence of tumors, etc.; or to some abnormal condition of the uterine supports. It is frequently caused by a vaginal cystocele or rectocele. Sudden prolapse is most apt to occur in an enlarged uterus or one unduly heavy by reason of tumors connected with it. It is most common in subinvolution after parturition.

Elevation of the uterus is produced by mechanical causes crowding or dragging it upward, as adhesions, tumors, etc. The vagina is drawn up and lengthened, and the vaginal portion of the cervix may be obliterated.

Inversion of the uterus consists of an invagination of the fundus. The fundus may be invaginated in the body, the fundus and body in the cervix, or the entire organ in the vagina. It usually occurs when uterine walls are relaxed, and is very frequently due to traction on placenta during parturition. It may take place spontaneously after p turition. It may be produced by intra-uterine tumors. The muc membrane of the inverted organ is frequently inflamed, particula when the inversion is complete.

Herniæ of the uterus are rare. *Ventral herniæ* may occur duri the latter months of pregnancy, the peritoneum, aponeuroses, and sk being forced outward to form a sac in which the uterus lies. *Crur herniæ* are produced by the drawing down of the uterus and ovaries in the sac of an intestinal hernia. *Inguinal hernia* may be produced the same way or be congenital. *Ischiatic hernia* has been seen. Pre nancy may occur in the uterus while situated in a crural or inguin hernia.

RUPTURE AND PERFORATION.

Rupture of the unimpregnated uterus is rare. It may, howeve occur when the uterine cavity is distended with blood or serum, or i connection with large myomata of the uterine walls.

In the gravid uterus, ruptures have been seen in nearly every mont of pregnancy, but most frequently toward the end. The rupture ma be produced by thinning of the uterine wall by tumors, or by violent contusions, or as the result of cicatricial contraction of the os.

The act of parturition is the most frequent cause. Malpositions o the fœtus, narrowing of the pelvis, protracted labor, thinning of th uterine wall from tumors, forcible use of the forceps and other instruments, are the ordinary causes. The rupture may be in the body of the uterus or the cervix, or both; it may be large or small; it may extend completely or only partly through the uterine wall. The consequences of partial rupture are hæmorrhage, gangrenous inflammation of the edges of the rupture, peritonitis, and usually death. In rare cases, the rupture cicatrizes and the patient recovers. Complete rupture usually causes death in a short time. The fœtus escapes partly or completely into the abdominal cavity. If the patient survives the immediate shock, fatal peritonitis soon ensues. In rare cases, the fœtus is shut in by adhesions and the patient survives.

Perforations of the uterus may be produced by carcinoma, by abscesses in its neighborhood, and by ovarian cysts.

HYPERÆMIA—UTERINE AND PERI-UTERINE HÆMORRHAGE.

Hyperæmia.—Aside from the active menstrual hyperæmia, the uterus may be hyperæmic in acute and chronic inflammation, as a result of displacement of the organ, and in certain forms of heart disease. The organ is usually enlarged, the mucous membrane swollen, and the veins more or less evidently dilated.

Hæmorrhage.—Effusion of blood into the cavity of the uterus occurs normally at the menstrual periods. For the abnormalities to which this function is subject, we refer to works on gynæcology. Effusions of blood at other than the menstrual periods may be caused by mechanical hyperæmia, by hæmorrhoids, by acute hyperæmia, by intra-uterine polypi and other tumors, by acute and chronic inflammation, by typhus fever, scurvy, etc., by ulcerating carcinoma, by abortions and miscarriages.

A peculiar form of hæmorrhage is the polypoid hæmatoma, or fibrinous polypus of the uterus. It occurs after parturition and after abortions. The portion of the uterine wall where the placenta was attached, with or without a portion of retained placenta, forms the point of attachment of the pedicle of the polypus. We find a large, polypoid, bloody mass firmly attached by a pedicle to the uterine wall. The uterus enlarges with the growth of the polypus; the cervix is dilated, and the thrombus projects into and may even fill up the vagina. The formation of such a thrombus is accompanied by repeated hæmorrhages.

Hæmorrhage in the substance of the uterus occurs in old age. The mucous membrane and uterine wall are infiltrated with blood, and there is some blood in the uterine cavity.

Peri-uterine or *Retro-uterine Hæmatocele* consists in an accumulation of blood around the uterus or in Douglas' cul-de-sac. It may consist of blood extravasated into the abdominal cavity, which settles into the pelvis; or, in consequence of local hyperæmia, there may be repeated extravasations of blood. In the latter case, the local peritonitis may produce false membranes, between the layers of which hæmorrhages take place. A similar condition rarely occurs in the male. The hæmorrhagic mass may become incapsulated, or may soften or suppurate and perforate into the rectum or vagina, or may be absorbed. A form of extra-peritoneal hæmatocele is described in which the blood lies between the folds of the broad ligament. The extravasation may proceed from hæmorrhage of any of the abdominal viscera or rupture of aneurisms; from vascular new-formed false membranes; from rupture of the varicose veins of the broad ligaments; from rupture of hæmorrhagic cysts of the ovaries; from the Fallopian tubes in tubal pregnancy or in hæmatometra; or from general causes, such as scurvy, purpura, etc. In some cases the extravasation begins at a menstrual period, and increases at the succeeding periods.

Ante-uterine Hæmatocele is of occasional occurrence, either in connection with the retro-uterine form or when the posterior cul-de-sac is obliterated.

INFLAMMATION.

I. *Of the Unimpregnated Uterus.*

Acute Catarrhal Endometritis.—In this disease, which in its grades may leave but little alteration after death, the mucous mem is swollen, hyperæmic, and sometimes the seat of punctate hæmor The epithelium may desquamate, and the mucosa contain an quantity of small spheroidal cells. The surface is more or less

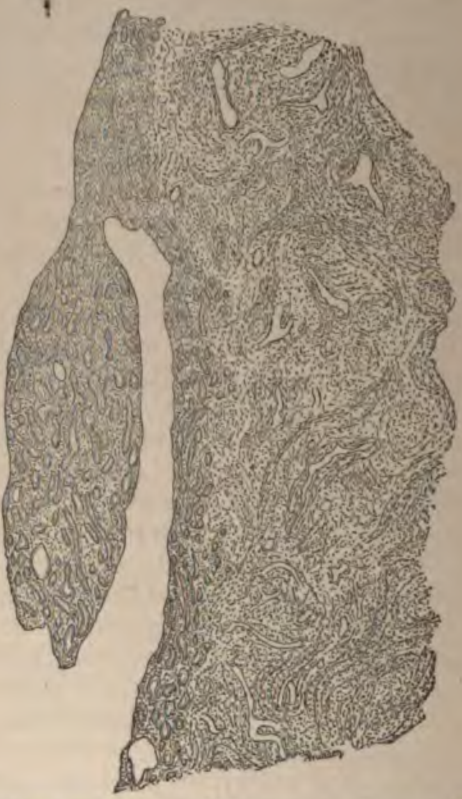

Fig. 185.—Chronic Endometritis with the Formation of a Polypoid Outgrowth Mucous Membrane.

covered with muco-purulent exudation. In severe cases, shreds cous membrane may be exfoliated. The lesion is usually most in the mucous membrane of the body, but may involve the cervix same time, or the cervix alone. The body of the uterus may be and hyperæmic. Acute catarrhal inflammation may be due to exposure during menstruation, the gonorrhœal infection, or accompany acute infectious diseases.

Chronic Endometritis.—This may be a continuation of an acute inflammation or begin as a chronic disease. In some of the lesser degrees of inflammation we find but slight changes after death. The mucous membrane, on the other hand, may be swollen, hyperæmic, and covered with muco-purulent exudation. In other cases, there is more or less well-marked thickening of the mucous membrane, which may present a smooth or a rough, papillary surface or polypoid outgrowths (Fig. 185). Owing to the hypertrophy of the uterine glands in this condition. these papillary outgrowths, which are not infrequently scraped off by the surgeon, often present the appearance of adenomata. Sometimes a thick layer of new-formed, very vascular tissue develops over the surface of the mucous membrane, largely covering in the uterine glands (Fig. 186). From the decomposition of extravasated blood in the mucous membrane, the latter may be mottled with brown or black. The glandular elements of the mucosa may be partially or almost entirely destroyed. The papillæ of the cervix may be hypertrophied, the mucous follicles swollen and their outlets obstructed, leading to the formation of the so-called ovula Nabothi. The uterine wall becomes flaccid and atrophied, or it may be hypertrophied, especially in the cervical portion. Ulceration of

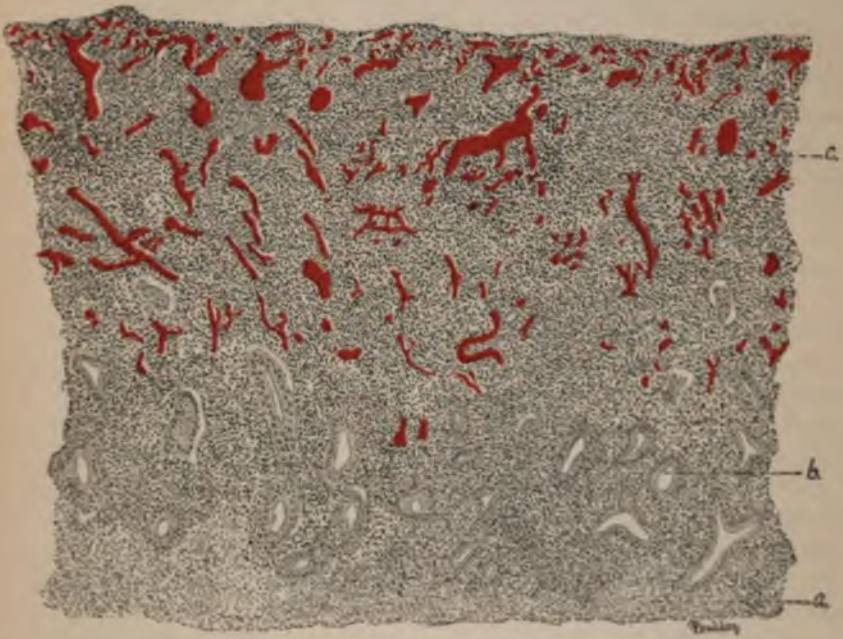

FIG. 186.—CHRONIC ENDOMETRITIS WITH THE FORMATION OF A THICK LAYER OF NEW-FORMED, VERY VASCULAR TISSUE OVER THE SURFACE OF THE MUCOUS MEMBRANE.

a, uterine muscle tissue; *b*, mucous membrane of uterus; *c*, new formed vascular tissue.

the mucous membrane, especially of the cervix, may occur. Contra tion or obliteration of the cervical canal may occur. The inflammatio may extend to the Fallopian tubes or to the vagina.

Chronic endometritis may exist at any age, but is most commo after puberty, and is produced by a great variety of causes. It ma occur in ill-nourished persons or in those suffering from exhaustin diseases. It may be due to displacements and tumors of the uterus, sub involution, injuries, etc.

Croupous Endometritis.—This form of inflammation is not very com mon. It occasionally occurs in the puerperal uterus, in acute infectiou diseases, cholera, typhoid fever, the exanthemata, etc. The diseas sometimes involves the vagina and Fallopian tubes. It may co-exis with croupous inflammation of the colon.

Tubercular Endometritis.—This usually occurs as part of a tubercu lar inflammation of the genito-urinary tract. We find a part or the whol of the cavity of the uterus lined with a rough, yellowish or gray, cheesy mass. which may deeply involve the muscular walls of the organ. At the edges of the ulcerating cheesy areas we may find well-defined miliary tubercles, or we may find tubercles scattered through the otherwise intact mucosa. The lesions resemble those of tubercular nephritis.

Syphilitic Endometritis.—The results of this infection are usually confined to the cervical portion, and consist of shallow or deep ulcerations and condylomata of the mucous membrane; or there may be a diffuse thickening of the mucosa.

Acute Metritis is usually the result of acute catarrhal endometritis. The organ is swollen, succulent, congested; the mucous membrane covered with muco-pus; the peritoneal coat congested. There may be small extravasations of blood in the wall or cavity of the uterus. The inflammation, in rare cases, becomes suppurative, and abscesses are formed in the uterine wall. These may perforate into the peritoneal cavity or into the rectum.

Chronic Metritis is the result of an acute metritis or accompanies acute or chronic endometritis, and is dependent upon the same conditions: subinvolution, displacements, tumors, active irritants, etc. The uterus is enlarged, the wall congested, thickened, and soft, or, owing to the new formation of connective tissue, hard and dense. The lesion may be most marked in the body or in the cervical portion.

Perimetritis.—The peritoneal coat of the uterus may be inflamed, with the production of membranous adhesions or of pus. The adhesions may be small or very extensive, and, owing to their contractions, may cause various distortions and displacements of the pelvic organs. The inflammation is usually an accompaniment of chronic metritis and endometritis. In prostitutes, such adhesions are of very common occurrence.

Parametritis.—The connective tissue about the uterus, between that organ and the reflexions of the peritoneum, may be the seat of suppurative inflammation. It most frequently causes the death of the patient, but may result in the formation of dense connective tissue about the uterus.

II. Of the Pregnant Uterus.

The forms of inflammation which have just been described may attack the pregnant uterus. Catarrhal endometritis may produce effusion of serum, extravasations of blood, and abortions. Metritis may lead to softening of the uterine wall, so that ruptures take place during labor. Perimetritis and parametritis produce adhesions and abscesses about the uterus.

Puerperal Inflammation.

For a week or more after delivery, we find the inner surface of the still dilated uterus rough, especially at the insertion of the placenta, and covered with blackened, gangrenous-looking shreds of blood, mucous membrane, and placenta. This condition is not to be mistaken for inflammation.

As a result of some injury to the uterus during or after delivery, and the action of some infectious material which may gain access to the tissues, the puerperal uterus is liable to become the seat of a series of severe and often destructive inflammatory and necrotic changes. These may be confined to the uterus; they may induce serious alterations in surrounding parts; they may lead to an involvement of the peritoneum, or to pyæmia and its accompanying lesions in the most distant parts of the body. In one series of cases, a more or less extensive gangrenous inflammation of the mucous membrane and the underlying parts may lead to the casting-off of larger and smaller shreds of necrotic tissue, and the formation of deep and spreading ulcers, which may be accompanied by severe parametritis and fatal peritonitis. This condition may be due to injury or to the presence of decomposing portions of retained placenta. In other cases the inflammation has a croupous character, which may affect the vagina and lead to necrosis and gangrene, ulceration, and peritonitis. In connection with either of the above forms of inflammation, or without them, there may be thrombosis of the uterine sinuses, purulent inflammation of the veins, suppuration and abscess in the uterine wall, and, owing to the generalization of the infectious material, to metastatic abscesses in the lungs, spleen, kidneys, etc. Or acute pleurisy, ulcerative endocarditis, purulent inflammation of the joints, hyperplastic swelling of the spleen and lymph nodes, may furnish characteristic features of the presence of an acute infectious disease. In some cases which rapidly pass to a fatal termination, the local lesions may be but slightly marked, and general alterations characteristic of pyæmia, such as metastatic abscesses, etc., be entirely wanting.

Bacteria, in the form of micrococci, are very constantly present i the exudation, in the lymph vessels, veins, and inflamed tissue of t uterus (see Fig. 187); often in enormous quantities in the peritone exudation and in the metastatic inflammatory foci. There is goo reason for believing that the destructive local processes are due to t presence and action of micrococci, and that the general infection in thi as in other forms of septicæmia and pyæmia, is dependent upon th same cause (see Septicæmia).

The Streptococcus pyogenes has been found in a considerable nu ber of cases, and is believed to induce the puerperal inflammation.

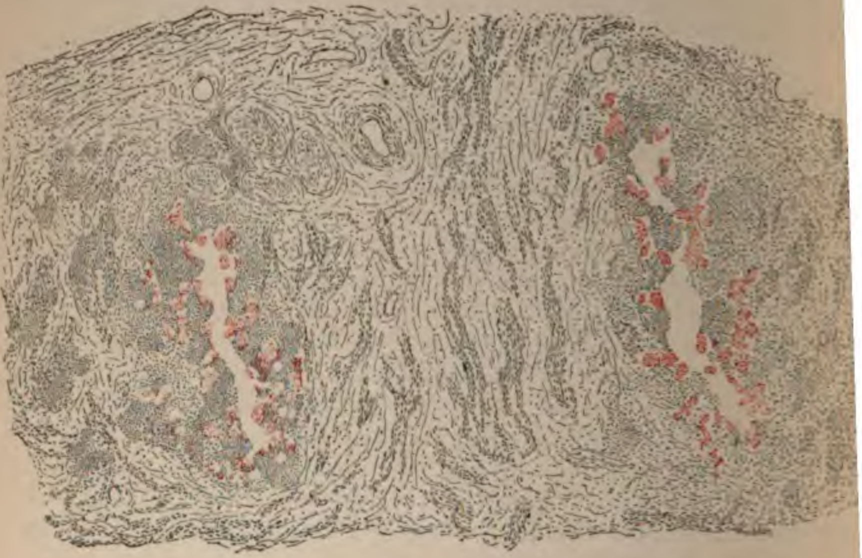

FIG. 187.—UTERINE PHLEBITIS FOLLOWING DELIVERY WITH RETAINED PLACENTA.
Death 9 days after delivery. Micrococci in the walls of the inflamed veins stained with fuchsin.

ULCERATION AND DEGENERATION.

Catarrhal, tubercular, and *syphilitic ulceration* have been mentioned above.

Phagedænic or Corroding Ulcer.—This rare form of ulceration usually occurs in old age, without assignable cause. It begins in the cervix and gradually extends until it may destroy the greater part of the uterus or even invade the bladder and rectum. The ulcer is of irregular form; its base is rough and blackish, its walls indurated. It should not be confounded with carcinomatous ulcer, which it considerably resembles.

Fatty Degeneration.—This may occur in connection with inflammatory changes, in acute infectious diseases, and in phosphorus poisoning.

Amyloid Degeneration in the uterus is of rare occurrence. It may affect the muscle fibres or the walls of the blood-vessels.

TUMORS.

Fibromata.—Dense nodular fibromata of the uterus are exceedingly rare, the so-called fibromata being in most cases myomata or fibro-myomata. *Fibroma papillare*, on the other hand, is a common form of growth from the mucous membrane. It consists of a more or less vascular connective-tissue stroma covered with epithelium. The surface may be smooth or villous. It may contain very numerous gland follicles, and then approaches the type of adenoma, or even carcinoma. The stroma may be loose and succulent, and resemble mucous tissue, forming the so-called *mucous polypi*; and these again may contain glandular structures. In any of these forms the blood-vessels may be abundant and dilated, forming telangiectatic or cavernous polypi. The adenomatous polypi may become cystic from the dilatation of the gland follicles.

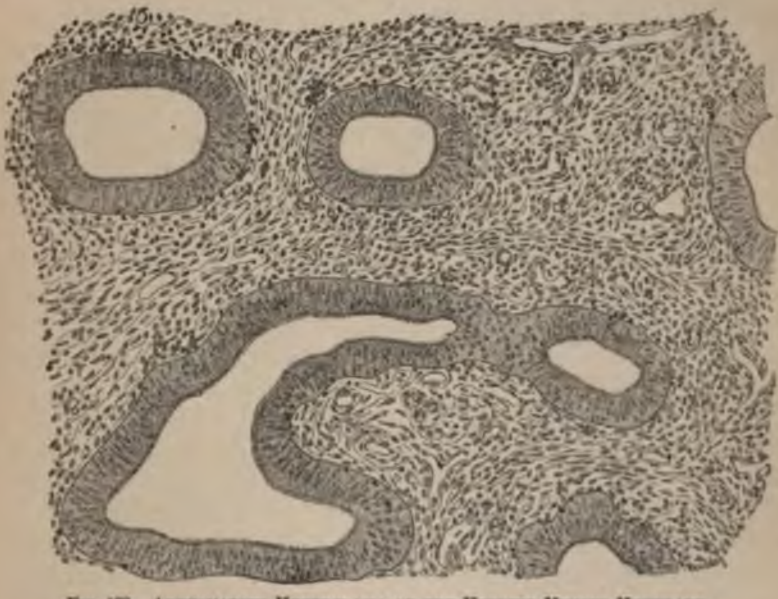

FIG. 188.—ADENOMATOUS HYPERPLASIA OF THE UTERINE MUCOUS MEMBRANE.
This section is from a large polypoid outgrowth which protruded from the cervix uteri.

Polypi of the uterus may be multiple or single, small or large. Numerous smaller and larger papillary outgrowths from the mucous membrane may occur in chronic endometritis. Single polypi may grow from the mucosa of the body of the uterus or from the cervix, and hang by a long pedicle down into the vagina.

The large number of glandular structures in many of these chron inflammatory, papillary, and polypoid outgrowths (Fig. 188) oft justify the name of adenomatous hyperplasia of the mucous membra or of adenomatous papillomata or polyps.

Syphilitic papillary growths in the form of pointed condylomata m form finely papillary, wart-like excrescences of variable size, particular on the cervix.

Myomata.—These tumors, whose characteristic structural elemen are smooth muscle cells (see Fig. 62), are the most common of uteri tumors and are frequently of no special practical importance, but a sometimes of very serious import. They are especially common in n groes. They are most frequently composed of both muscular and fibro tissue—fibro-myomata—but the relative amount of the two kinds tissue is subject to great variation. They are most apt to occur aft puberty, and usually in advanced life. They may be single or multipl small or of enormous size; are usually sharply circumscribed, whitish pink, dense and hard, or sometimes soft, and present on section inte lacing bands or irregular masses of glistening tissue. Their favorit situation is in connection with the body of the uterus, but they ma occur in the cervix or in the folds of the broad ligaments. Accordin to their position, we may distinguish subserous, submucous, and intra parietal forms. The *subserous myomata* grow from the outer muscula layers of the uterus in the form of little nodules. As they increase i size they may become separated from the uterine wall and remain attache only by a narrow pedicle or by a little connective tissue. They may wor their way between the folds of the broad ligament until they are at som distance from their point of origin. Some authors mention cases i which the tumors became entirely detached from the uterus and wer free in the abdominal cavity. In some cases, the tumors excite inflam mation of the adjacent peritoneum, leading to the formation of adhesion or of collections of pus. Cases are recorded in which, owing to th atrophy of the pedicle, subserous myomata have become completely detached from the uterus and were held in place and nourished by peritoneal adhesions. In other cases, the tumor reaches a large size, but remains firmly attached to the uterus. This organ may then be drawn upward, the cervix and vagina being elongated and narrowed. The traction may be so great that the body of the uterus is entirely separated from the cervix. The bladder may also be drawn upward, producing incontinence of urine and cystitis. Subserous myomata are very often multiple and frequently attain great size.

The Submucous Myomata grow from the inner muscular layers of the uterine wall. They commence as rounded nodules which lift up the mucous membrane. Their usual position is the fundus uteri. They rarely occur in the cervix. As the tumors increase in size they project into the uterine cavity. They then remain continuous with the uterine wall over a large area, or are attached by a large or small pedicle. They are usually well supplied with vessels. The uterus dilates with the growth of the tumor, and its walls may be also thickened. The tumor may reach such a size as to entirely fill the cavity of the dilated uterus and project through the cervix into the vagina.

The submucous myomata are usually single, although there may be at the same time subserous and intraparietal tumors. They are frequently soft. If they are of large size and polypoid in form, they may project through the cervix and drag down the fundus of the uterus, producing inversion. The mucous membrane covering them may be atrophied or hyperæmic, with dilated blood-vessels, and may thus give rise to severe and repeated hæmorrhages. Hæmorrhage may occur in the substance of these tumors. Inflammation, suppuration, and gangrene may also occur. The surface may be ulcerated. In some cases the pedicle of the tumor is destroyed, and it is spontaneously expelled.

The *intraparietal myomata* grow in the substance of the uterine wall, but, if they attain a large size, project beneath the serous or the mucous coat. They are found in every part of the uterus, but are most frequent in the posterior wall.

The shape of the uterus is altered in a great variety of ways by the presence of these tumors; its cavity is narrowed, dilated, or misshapen; it undergoes flexion and version in every direction. The tumors may sink downward and become attached to the posterior wall of the vagina, looking as if they grew from it. They may, without the formation of a pedicle, project into the cavity of the uterus, fill it up, and project through the cervix. The uterus is dilated, its wall hypertrophied or atrophied.

The tumors themselves may undergo a variety of secondary alterations. The muscle fibres may undergo *fatty degeneration*, and the tumor diminish in size, or even undergo, it is said, entire destruction and atrophy. *Calcification* may occur, converting a part or the whole of the tumor into a stony mass. The intraparietal and submucous myomata may give rise to profuse hæmorrhages; they may suppurate and become gangrenous.

Sometimes the tumors or circumscribed portions of them are very vascular, constituting the *telangiectatic* or *cavernous* variety. These tumors, which possess some of the characters of erectile tissue, may suddenly change in size from a variation in the amount of blood which they contain.

A very important change which is sometimes found in these tumors is the development of cysts in their interior (fibro-cystic tumors). This sometimes takes place in those tumors which grow outward beneath the peritoneal coat. We find one or more cavities communicating with each other, with rough, trabeculated walls. The appearance is more tha cavities than of cysts. There may be a number of smaller cavities, they may fuse to form one large one. The fluid contained in the c ties is like synovia, or is mixed with blood. These cystic myom may reach an immense size and fill the abdominal cavity. The di nosis, during life, between them and ovarian cysts is often very diffic and they have frequently been the subjects of fatal operations.

In the cervix uteri, myomata are rare. They may grow as pol beneath the mucous coat, or produce enlargement of the anterior or p terior lips, or may grow outward into the abdominal cavity.

Combinations of myoma and sarcoma sometimes occur—*myo-s coma.*

Sarcomata may occur as primary tumors in the mucous membrane the uterus, either in the form of a diffuse infiltration or as a circu scribed nodular or polypoid mass. They frequently involve the mus lar wall, are liable to hæmorrhage and gangrene, and, particularly the diffuse form, are liable to recur after removal. They may cons largely of spindle or spheroidal cells, or both. It is said that sarcoma the uterus is more liable to occur at an advanced age than at an ea period, as is the rule with sarcomata of other organs.

Angioma.—A small, cavernous angioma of the wall of the uterus h been described.

Carcinoma.—The carcinomata of the uterus commence most fr quently in the cervix and portio vaginalis, and the most common for is the epithelioma. The growth of epitheliomata of the cervix ute proceeds under three tolerably distinct forms, which, however, fr quently merge into one another.

1. *The Flat, Ulcerating Epithelioma.*—This form of cancer commenc as a somewhat elevated, flat induration of the superficial layers of th cervix, sometimes circumscribed, sometimes diffuse. This inductio is due to the growth of plugs and irregular masses of epithelial cell into the underlying tissue. Ulceration usually commences early, an may proceed slowly or rapidly. The edges of the ulcer are irregular indurated, and somewhat elevated. The ulceration of the new-forme cancerous tissue at the edges is usually progressive, so that the vagina portion of the cervix, the cervical canal, the vagina, and even the bladder and rectum may be involved. More or less extensive hæmorrhage and necrosis of the base of the ulcer are liable to occur. The entire cervix may be destroyed.

2. In another class of cases, the carcinomatous growth develops under the form of papillary or fungous excrescences, which may form larger or smaller masses composed of epitheliomatous tissue. Hand-in-hand with this projecting growth there may occur an epithelial infiltration of the underlying tissue of the cervix. These growths are often

quite vascular, and may give rise to severe hæmorrhages. They may ulcerate, and thus produce great destruction of tissue.

3. In still another class of cases, there is a more or less deep infiltration of the submucous tissue, either diffuse or in circumscribed nodules, with epithelial cell masses. We find at first, in the vaginal portion of the cervix, in the submucous connective tissue, either nodules or a general infiltration of a whitish new growth. The cervix then appears large and hard. Very soon the mucous membrane over the new growth degenerates and falls off; the superficial layers of the new growth undergo the same changes. After this, the formation of the new growth and its ulceration go on simultaneously, producing first an infiltration and then destruction of the cervix, and often of a part of the body of the uterus. The growth frequently extends to the vagina, the bladder, and rectum with the same destructive character, so that we often find the cervix and upper part of the vagina destroyed, and in their place a large cavity with ragged, gangrenous, cancerous walls (Fig. 189). Less frequently the pelvic bones are invaded in the same way. Not infrequently the ureters are surrounded and compressed by the new growth, so that they become dilated. The dilatation may extend to the pelves and calyces

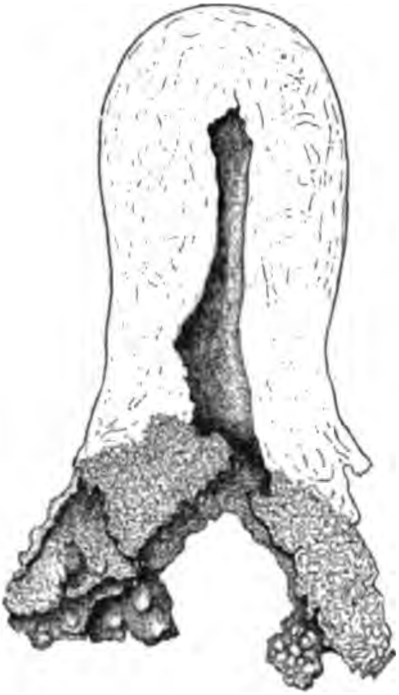

Fig. 189.—Carcinoma of the Cervix Uteri (Ulcerating).

of the kidneys. The new growth may begin in the cervix, and exte uniformly over the internal surface of the cervix and of the body the uterus. The entire uterus is converted into a large sac, of whi the walls are infiltrated with the new growth, while the internal surf is ulcerating and gangrenous. In some cases there is a considera formation of new, dense connective tissue, so that the growth has a sc rhous form.

In rare cases, the growth begins in the upper part of the cervix or the body of the uterus, while the lower part of the cervix is not involv In all of these cases, the epithelial cells of the new growth follow m or less closely the type of the epithelial cells of the part from which th spring.

In still another class of cases, in which the new growth may be in t form of nodules, or diffuse infiltrations, or polypoid masses, or may p sent more or less extensive alterations, the cells are irregular, polyhed in shape, the tumor belonging to the class of glandular or medull carcinomata. These also usually commence in the cervix and, accordi to the views of many writers, probably in the mucous glands.

In rare cases the entire wall of the uterus is infiltrated with the n growth, and the organ is much enlarged. *Colloid carcinoma* someti occurs, but is rare.

While we may for convenience recognize the above types of carcino of the uterus, it should be borne in mind, as above stated, that they not apt to be perfectly distinct, and some of them may merge into another or exist simultaneously. Exudative inflammation is of frequ occurrence in these as in other tumors of the uterus (see Fig. 72).

As a result of the ulceration of these various forms of carcinon recto-vaginal fistulæ may be formed; the lumbar lymph nodes may involved, and metastases in distant organs are occasionally though frequently formed. Frequent and profuse hæmorrhages, gangren destruction of tissue, the absorption of deleterious materials, etc., apt to lead to the development of a more or less profound anæmia a cachexia.

PARASITES AND CYSTS.

Various forms of *bacteria* are of frequent occurrence. *Echinococ* has been found in the body and neck of the uterus, and may rupture i the peritoneal cavity or into the vagina.

Cysts.—Aside from the cysts which develop in tumors of the uter in the cervix uteri the mucous follicles are frequently so dilated as form cysts filled with a gelatinous material and more or less epitheliu These cysts may be large or small, and are frequently called *ovula Na thi.* Sometimes there is an inflammatory growth of new connective t sue about these cysts. In other cases the cysts may project from t mucous membrane in the form of polypi. Similar changes are infrequently found in the body of the uterus from the dilatation of occluded uterine glands. Dermoid cysts are rarely found in the walls of the uterus.

THE OVARIES.

MALFORMATIONS.

One or both ovaries may be absent, the other organs of generation being also absent or undeveloped. Or the ovaries may be only partially developed. Absence or arrest of development of one ovary is sometimes met with in otherwise well-formed individuals. It is sometimes accompanied by a low position of the kidney on the same side. The ovaries may pass into the inguinal canal or into the labia majora, and remain fixed there through life. Less frequently they are found in the crural canal or the foramen ovale.

CHANGES IN SIZE.

The ovaries may become larger than normal by chronic inflammation, by the formation of cysts and tumors. They may become atrophied in old age, the Graafian follicles disappearing, and the organ shrivelling into a small, irregular, fibrous body. Atrophy may be produced by ascites, by chronic inflammation, or from unknown causes. As the result of the maturing and rupture of the Graafian follicles, with and without pregnancy, the surface of the ovary, which before puberty is smooth, may become roughened by irregular cicatricial depressions.

CHANGES IN POSITION.

In adult life, the ovaries may pass as herniæ into the inguinal or crural canal, the foramen ovale, or the umbilicus.

The position of the ovaries in the abdomen may be changed by the pressure of tumors, the traction of false membranes, etc. It may occur in enlarged ovaries or in those of normal size, and by the compression of the veins may lead to congestion and chronic inflammation of the organ.

HYPERÆMIA AND HÆMORRHAGE.

Aside from the normal hyperæmia of the ovaries during menstruation, the vessels may be congested in inflammation, in displacements with interference with the venous circulation, in certain diseases of the heart, etc., and may then be followed by chronic inflammation.

The menstrual periods are accompanied by the effusion of blood into a Graafian follicle. Normally the amount of blood is small, becomes solid, is decolorized, and then gradually absorbed. Sometimes the effusion of blood is much greater; the follicle filled with blood is as large as a pigeon's egg. The blood may remain in the follicle and be absorbed.

peral condition, either as part of a general peritonitis or as a
affection.

With puerperal peritonitis, both ovaries are usually inflam
are swollen, congested, soft, infiltrated with serum or pus, or
ous. The inflammation may attack principally the capsule, th

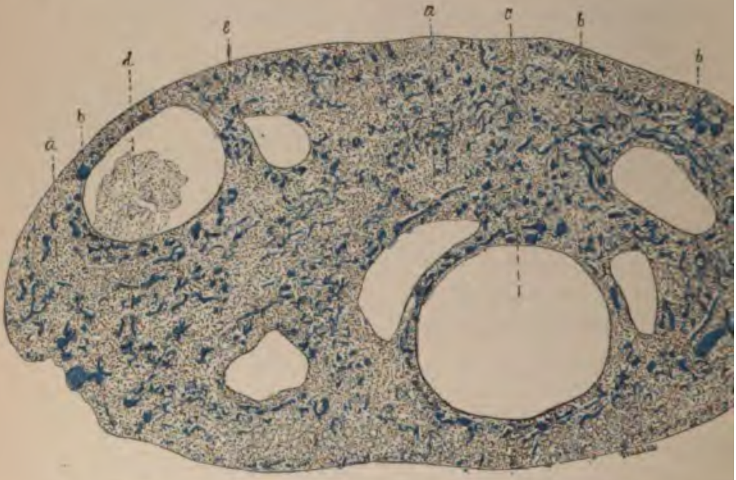

FIG. 190.—CHRONIC OOPHORITIS WITH DILATED BLOOD-VESSELS AND CYSTS.

a, dense connective-tissue stroma; *b*, dilated veins; *c*, cysts; *d*, cyst with granul
e, cortical zone of immature Graafian follicles.

or the follicles. Inflammation of the capsule results in adhe
collections of pus shut in by false membranes; of the strom

Inflammation of the ovaries unconnected with the puerperal condition is not common, but it may occur in connection with acute or chronic peritonitis or perimetritis. It is usually confined to one ovary. Either the follicles, stroma, or capsule, or all together, may be involved. The inflamed follicles are enlarged, their walls thickened; they may contain bloody or purulent fluid. The stroma becomes infiltrated with serum or pus, and later we may find abscesses or fibrous induration of the organ. The inflammation of the capsule may lead to the formation of membranous adhesions between the ovary, Fallopian tube, and surrounding parts.

Chronic Interstitial Oöphoritis is not infrequently preceded by an acute inflammation, or it may gradually develop as an independent condition, often determined by some mechanical interference with the blood-current. The organ may be increased in size, owing to the formation of loose cellular, or of dense, firm, new connective tissue. Under these conditions, the blood-vessels, especially the veins, may be widely dilated and cysts in varying number and size may be present (Fig. 190). Sometimes the new-formed dense connective tissue may be largely limited to the surface of the organ, so that the albuginea may become so dense and thick that the functions of the organ must, as it would seem, be permanently interfered with. Under these conditions the surface of the ovary may be smooth or rough.

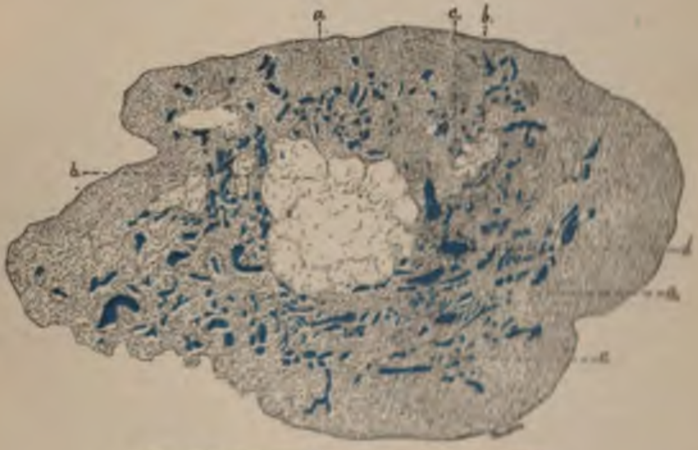

FIG. 191.—CHRONIC OÖPHORITIS WITH ATROPHY.

From a case of valvular disease of the heart with chronic metritis and endometritis. *a*, thickened and dense interstitial tissue; *b*, old corpora lutea; *c*, arteries with greatly thickened walls; *d*, dilated veins.

On the other hand, the organ may be smaller than normal as the result of the formation of dense new interstitial connective tissue, and its surface greatly roughened and distorted. Sometimes the formation of new dense tissue may be largely confined to the walls of the arteri which become prominent and tortuous. The atrophied ovary may largely made up of thick-walled arteries and fibrous masses which the result of incomplete resolution of the corpora lutea (Fig. 191).

Tubercular Inflammation of the ovaries is rare, and may accompa tubercular inflammation of other organs, particularly the peritone and Fallopian tubes. It usually results in the production of che nodules of considerable size.

Syphilitic Inflammation in the form of gummata is uncommon.

TUMORS.

Fibromata.—These tumors are not very common nor usually of gre importance. They may be very small or of great size. They are usual dense in texture, and in a considerable number of cases seem to origina in the tissue formed in the closure of the ruptured Graafian follicl They may contain cysts or be accompanied by cysts of the surroundi stroma. Papillary fibromata of the surface of the ovary are sometim seen.

Leiomyomata containing more or less fibrous tissue are of occasion occurrence.

Sarcoma of the ovaries is not common. It is usually primary, b may be metastatic. It is usually of the spindle-celled variety, but m contain areas of spheroidal-celled tissue or more or less fibrous tissu The tumors may be hard or soft, and are apt to involve both ovaries.

Chondroma of the ovaries is described, but is rare; cartilage not i frequently occurs, however, in dermoid cysts.

Carcinoma, usually of the glandular variety, may occur as a primar tumor of the ovary. It may be due to a continuous infection fro neighboring organs, or more rarely it is of metastatic origin. Althoug the glandular medullary carcinomata are the most common, scirrhou melanotic, and colloid cancer sometimes occur. Some forms of carc noma stand in very close relation with certain of the cystic adenomat (see below).

Adenomata (Cystic Adenomata; Compound Ovarian Cysts).—The growths, which may occur in one or both ovaries, form one of the mos common and important classes of ovarian tumors. Some of their mos noteworthy and important features depend upon their tendency to th formation of cysts. It should be remembered, however, that the primary lesion is a true new formation of glandular tissue, and not, as i the case of most cysts, a transformation, by retention or otherwise, o pre-existing structures.

The growth primarily consists of a fibrous stroma, in which ar tubular follicles lined with cylindrical epithelium. Or, in some cases, i consists of the above follicular or glandular structures associated wit papillary outgrowths from a fibrous stroma, which are covered with cylindrical epithelium. Sometimes one, sometimes the other form of growth—the glandular or the papillary—predominates (Fig. 192). There is, as above stated, a marked tendency, particularly in the glandular form of adenoma, to a dilatation of the follicles by a semi-fluid material, and the formation of cysts. There may be a number of follicles equally dilated, so as to form a number of cysts of moderate size; or only a few follicles are enormously dilated to form a large *multilocular cyst* with but few compartments. The walls of the cysts may fuse together, and be absorbed so as to form one large cyst divided by incomplete septa —*unilocular cysts*. The stroma in which the follicles and cysts are imbedded may be largely developed or very scanty.

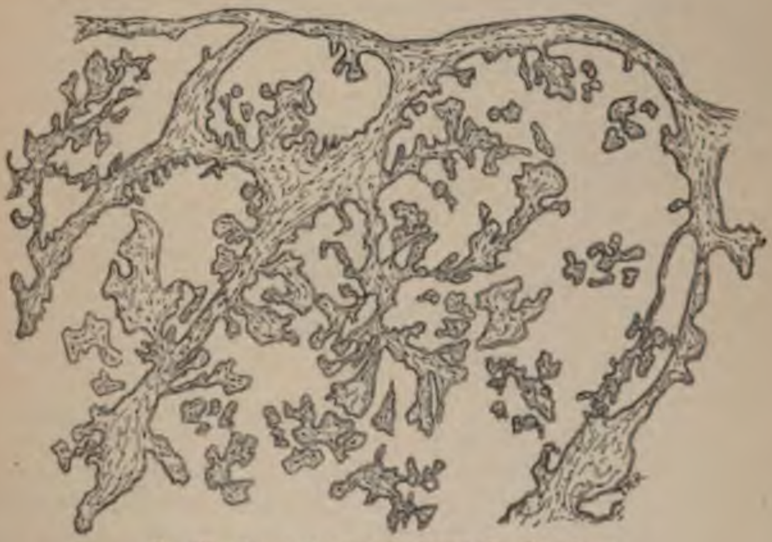

FIG. 192.—CYSTIC ADENOMA OF OVARY (Papillary form).

The walls of the larger cysts are composed of fibrous tissue which is dense in the outer layers, more cellular in the inner, upon which the epithelium is placed. They may be thin and membranous, or we find developed on their internal surfaces an intracystic growth composed of a fibrous stroma and tubular follicles. These secondary follicles may also be filled with fluid and form larger and smaller cysts. The intracystic growths may be so large as to fill up the original cysts. Sometimes the intracystic growth presents very little dilatation of its follicles, so that the entire tumor has more the character of a solid growth than of a cyst.

In many of the cysts in which the formation of follicles and their dilatation are well shown, the growth of the papillomatous projections is also seen, so that the cyst cavity may be more or less filled with cauliflower-like tufts.

The cylindrical epithelium lining the cysts usually forms a sin layer, but, owing to the accumulation of fluid, the cells may become fl tened and atrophied, or they may be fatty or desquamated. The co tents of the cysts differ considerably in different cases, and even in d ferent cysts in the same case. It may be tough and ropy, or gelatin or serous; transparent and colorless, or yellow or reddish, or redd brown; or it may be turbid and colorless, or variously colored, re brown, or chocolate.

Chemically the cyst contents, when thick and ropy, contain mucin paralbumen, and perhaps other less well-known compounds belonging the same class. It is believed that the peculiar ropy character whi the fluid often possesses is due to the paralbumen, but the chemical n ture and relations of this substance are still matters of dispute. It probable that the contents of these cysts are, so far as the mucin a paralbumen are concerned, produced by a metamorphosis of the prot plasm of the lining cells, similar to that by which the mucin is produc in the mucous glands and in mucous membranes. We frequently fi the cylindrical cells presenting the form of the so-called "beaker cells

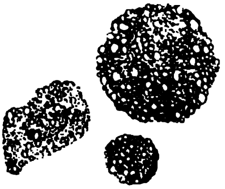

Fig. 193.—Cells from Contents of an Ovarian Cyst in a Condition of Fatty Degeneratio

and in some cases the mucous contents of the cysts are seen to be con tinuous with the similar contents of the beaker cells. It is probabl that much of the fluid contents of the cysts comes from simple transud tion.

Microscopically the contents of these cysts present also considerabl variation. We may find almost no structural elements; or there may b red blood-cells in variable quantity, and pus cells in various stages o granular or fatty degeneration or of disintegration, so that variousl shaped fragments of the cells appear. Then we may find cylindrical, o flattened, or polyhedral cells, either well preserved, swollen, or in a stat of fatty degeneration (Fig. 193), or we may find fragments of these cells It is these various forms of cells, often more or less swollen and in a con dition of more or less well-marked granular and fatty degeneration, whic have been considered characteristic of the ovarian cysts and are sometimes called *Drysdale's* corpuscles. While, however, they are of frequent occurrence under these conditions, they are by no means pathognomonic, since we find them in the contents of various kinds of cyst and cavities where the cells are undergoing degeneration. In addition to the above structural elements, we may find free fat-droplets, cholestearin crystals, pigment granules, and more or less granular detritus. The material filling these cysts is sometimes called colloid, and the cysts are frequently called colloid cysts; but we believe that the above view of their nature is the correct one.

Numerous secondary changes are liable to occur in these cysts. The cells may become fatty and peel off, so that we may find in some parts only a connective-tissue wall. The walls may atrophy, may become infiltrated with salts of lime, or contain concentrically lamellated lime concretions. Inflammatory changes may occur in them. There may be a suppurative inflammation of the walls leading to the formation of abscesses, or pus may be mingled with the cyst contents; the epithelium may be exfoliated and granulation tissue may form in the walls. Chronic inflammation may lead to considerable thickening of the walls, and to adhesions with neighboring parts. Hæmorrhages, sometimes very extensive, may occur in inflammation, or as the result of other disturbances of the circulation, so that some of the cysts may be filled with blood. Inflammatory softening, gangrene, etc., of the walls may lead to perforation, so that the contents of the cysts may be discharged into the peritoneal cavity, or, in virtue of adhesions, into the bladder, vagina, or rectum. Perforation may be caused by the piercing of the walls by the papillomatous growth. Carcinoma may be developed from the epithelium of the cysts. Since these cysts sometimes reach a very large size, they may produce the greatest variety of disturbances in the abdominal cavity, which need not be enumerated here.

They probably originate in the glandular epithelium of the ovary either before or after the formation of the Graafian follicles.[1]

Follicular Cysts of the Ovary.—The Graafian follicles may be dilated so as to form cysts. This may occur in one or both ovaries, and the cysts may be small or large, single or multiple. They are usually found after middle life, but may occur during youth, childhood, or even in the fœtus. The follicles dilate from the accumulation of fluid within them; the ovum is destroyed, the epithelium flattened. The contents are usually serous and colorless, but may be viscid, turbid, purulent, or variously colored, red, yellow, or brown. The ovary may be crowded with numerous cysts of moderate size, whose adjacent walls may coalesce and atrophy, forming communications between them.

A variety of this form of cyst is formed by the dilatation of a follicle containing a corpus luteum. Such cysts may communicate with a Fa lopian tube.

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[1] For more extended descriptions of the cyst adenomata of the ovaries, see *Waldeyer*, "Die epithelialen Eierstöcksgeschwülste," Archiv für Gynäkologie, Bd. 1, Heft 2, pp. 252–316, 1870. Also *Klebs*, "Handbuch der pathologischen Anatomie," vierte Lieferung, p. 796, 1873.

Dermoid Cysts.—These cysts may be uni- or multilocular, are usual of moderate size, but sometimes become as large as a man's head larger. Their fibrous walls may be thick or thin, and portions of t internal surface may present more or less completely developed cuticul structures, such as corium, papillæ, epidermis, hairs and hair follicl sebaceous glands, etc. The cavity may contain a thick, whitish, grea material composed of flattened epithelium, fat, or cholestearin crysta Or the cavity or walls may contain masses of irregularly formed ha teeth, bone, cartilage, striated muscle, and nerve fibres and cells. Su growths, which are doubtless of embryonal origin, may exist for ma years without causing inconvenience; but inflammatory changes m occur in them, leading to adhesions and perforations into adjacent orga They may form the nidus for the development of carcinoma, or th may calcify.

In addition to the above-described adenoid, dermoid, and simp follicular cysts, there are a number of composite forms of not infreque occurrence. Thus, in connection with dermoid cysts or separately, find larger and smaller cysts lined with ciliated epithelium. Then the are several cases described of cysts which partake of the characters both adenoid and dermoid cysts. Such cysts may be multilocular a be lined with flattened, cylindrical, or ciliated epithelium, and may co tain epidermal cells, cholestearin or mucin, etc.[1]

Small cysts, sometimes pediculated, sometimes not, of doubtf origin and usually of no special significance, are frequently found gro ing from the broad ligament near the ovary. The walls are usually ve thin, lined with flattened epithelial cells, and the contents serous.

Cysts of the Parovarium, lying between the peritoneal layers of t broad ligament, are usually small, but may be as large as a man's hea They are usually lined with ciliated epithelium, but sometimes with fla tened non-ciliated cells. The contents may be serous or may be thic and contain mucin and paralbumen.

THE FALLOPIAN TUBES.

MALFORMATIONS.

Absence of both tubes occurs with absence of the uterus. One tu may be absent, with arrested development of the corresponding side the uterus. Both tubes may be imperfectly developed; either of the ends may be closed; they may be inserted into the uterus at an abnormal place; they may terminate in two or three abdominal ostia.

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[1] A tolerably full bibliography of the tumors of the ovaries may be found *Tait's* "Pathology and Treatment of Diseases of the Ovaries," 4th ed., Wm. Wood Co., p. 131.

CHANGES IN POSITION AND SIZE.

The Fallopian tubes may participate in the various malpositions of the uterus and ovaries; but they are most frequently displaced by the contraction of adhesions formed in perimetritic and periovarial inflammations.

The lumen of the tube may be partially or completely closed as the result of inflammation of the mucous membrane; of peritonitis about the fimbriated extremity; of tumors or inflammation of the uterus; or by pressure from without, as by adhesions, tumors, etc. It may become stopped by plugs of mucus or pus.

P. K. W.

Dilatation of the tubes may be produced by an accumulation of catarrhal or other exudation, when there is partial or complete stenosis at some portion of the tube. The dilatation may be moderate, converting the tube into a tortuous, sacculated canal containing mucous or serous fluid; or, more rarely, large cysts may form containing several pounds of serous fluid—*hydro-salpinx*. As the fluid collects, the epithelium may become flattened or fatty or may desquamate. As a result of an inflammation in the walls of the dilated tube, the contents may be mixed with pus or blood. Rupture of a dilated tube sometimes occurs; or severe and even fatal hæmorrhage may take place into its cavity. Papillary growths are sometimes found springing from the inner wall of the cysts.

HÆMORRHAGE.

Hæmorrhage into the tube may occur in puerperal women with retroversion of the uterus, with abortions; hæmatometra and tubal pregnancy; in acute infectious diseases. The blood may undergo degenerative changes and be largely absorbed, or it may escape into the peritoneal cavity and cause peritonitis.

INFLAMMATION (SALPINGITIS).

Catarrhal Inflammation of the mucous membrane of the Fallopian tubes commonly occurs in connection with endometritis, frequently in the puerperal condition. In the acute stage, the mucous membrane is hyperæmic and swollen, and covered with a muco-purulent exudation. The inflammation may subside, leaving no lesions, but it more frequently becomes chronic, and may then result in peritoneal adhesions, obliteration of the tubes, and dilatation.

Suppurative Salpingitis.—This inflammation of the mucous membrane may assume a suppurative character, particularly in connection with puerperal metritis and peritonitis, but sometimes as a result of gonorrhœal inflammation.

Under these conditions, the wall of the tube may be involved, and pus may exude from the abdominal ends. It is difficult, in many cases of suppurative salpingitis associated with peritonitis, to say which is the primary lesion.

In some cases, there is a considerable collection of pus in the tubes, causing dilatation—*pyo-salpinx.* These collections may rupture into the peritoneal cavity, or the pus may escape into a cavity shut in by adhesions, or may perforate into the intestine or bladder. Or it may dry and finally become calcified.

Tubercular Inflammation.—This form of inflammation in the tubes is most frequently seen in its later stages, when the mucous membrane is partially or entirely converted into a thick, cheesy, often ulcerating layer. The lumen of the tubes may be dilated, the walls thickened from chronic inflammation. This lesion may occur by itself, or may be associated with tubercular inflammation of the lungs, or of the other genito-urinary organs, or the peritoneum. The lesion usually commences at the abdominal ends of the tubes, and both tubes are apt to be involved.

Syphilitic Inflammation in the form of a diffuse thickening of the wall by gummatous tissue has been described.

TUMORS.

Small *fibromata* and *fibro-myomata* sometimes occur in the wall of the tubes or in the fimbriæ. Small *lipomata* have been seen between the folds of the broad ligament in close connection with the tubes.

Carcinoma of the tubes is usually, if not always, secondary to cancer of the uterus or ovaries.

Cysts, usually of small size, sometimes pediculated and with thin walls, are frequently seen in the peritoneal covering of the tubes or in the fimbriæ. They are believed to be of embryonal origin.

EXTRA-UTERINE PREGNANCY.

Tubal Pregnancy.—The impregnated ovum is in some way hindered from passing into the uterus, becomes fixed in the tube, and is there developed. The villi of the chorion grow into the mucous membrane of the tube, forming an incomplete placenta. Rare cases are recorded in which the placenta was situated in the uterus while the fœtus was developed in the tube. The embryo and its membranes are developed until they reach such a size that the tube surrounding them ruptures. This may occur in the first month or not until much later. In rare cases, when the wall of the tube was extensively involved in the formation of the placenta, the development has gone on until term. The ovum may remain in the tube after the rupture; or may escape into the peritoneal cavity, still enveloped in its membranes; or the membranes may be ruptured and left in the tube. The rupture is generally attended with fatal hæmorrhage. In some cases, death is caused by the rupture of a dilated vein while the tube is still intact. Hæmorrhage into the sac may occur before its rupture.

In rare cases, death does not take place, and the fœtus is shut in by adhesions and false membranes. The embryo soon dies. In favorable cases, there is a slow absorption of the soft parts of the fœtus, the bones are separated and left imbedded in a mass of fibrous tissue, fat, cholestearin, and pigment. Or the fœtus retains its shape and becomes mummified, and may then be incrusted with the salts of lime.

In unfavorable cases, degeneration and gangrene of the fœtus take place rapidly, with inflammation and suppuration of the surrounding tissues. There may be perforation and escape of the broken-down fœtus through the rectum, vagina, bladder, or abdominal wall. The patient may die from peritonitis or exhaustion, or may recover after the escape of the fœtus.

In some cases, the fœtus may escape through a rupture of the tube into the space between the folds of the broad ligament.

Tubo-abdominal Pregnancy is produced by the development of the ovum in the fimbriated extremity of the Fallopian tube Adhesions are formed, so that the fœtus is partly in the end of the tube and partly in the abdomen.

Interstitial Pregnancy.—The ovum in these cases is arrested and developed in the portion of the tube which passes through the wall of the uterus.

Abdominal Pregnancy.—The ovum, after escaping from the ovary, does not enter the Fallopian tube, but becomes fixed to the peritoneum, usually at some part near the ovary. It is surrounded by thickened peritoneum and develops in that position.

Ovarian Pregnancy.—The existence of this form of pregnancy is doubtful and difficult to prove, but there are some cases in which it seems probable that the ovum develops in its Graafian follicle. The placenta may be attached to the tube or to the abdominal wall.

In all forms of extra-uterine pregnancy, the uterus becomes enlarged, and a sort of decidua is formed on its internal surface.

LESIONS OF THE PLACENTA.

Aside from the variations from the normal in size, shape, and position, for a description of which we refer to the works on obstetrics, we may briefly mention here some of the more important structural changes which the placenta may undergo.

Hæmorrhage.—This may occur either on the maternal surface in the decidua; or between the fœtal surface and the membranes; or in the substance of the placenta. The latter form of hæmorrhage constitutes the true *placental apoplexy.* This may occur as the result of rupture of a placental sinus. The placental tissue is crowded apart, and a blood-clot, often infiltrating the parenchyma, is formed. This may lead to abortion, or the blood may undergo disintegration and absorption, and its place be occupied by a cicatrix. The placental tissue in its vicinity may undergo fatty degeneration. Under other conditions, without evidence of rupture of the vessels, the placental tissue may become infiltrated with blood in the form of an infarction. In this, degenerative changes, similar to the above, may occur, leading to fibrous induration of the placenta.

INFLAMMATION (PLACENTITIS).

Suppurative Inflammation of the placenta, with the formation of abscesses, is of rare occurrence as the result of injury.

Chronic Indurative Inflammation of the placenta may result in the formation of circumscribed masses of cellular and loose, or dense and cicatricial connective tissue, or in a diffuse formation of connective tissue, which may interfere with the nutrition of the fœtus and cause abortion. The new-formed connective tissue may undergo fatty degeneration or calcification.

In another class of cases, the new connective tissue is formed mainly in the walls of the vessels, particularly the arteries. This may occur in circumscribed portions of the vessels, leading to nodular growths around the arteries, or it may occur extensively along the various ramifications of the vessels, converting them into thick fibrous cords. The change is primarily in the adventitia, but all the coats of the vessel may become involved, leading to more or less complete obliteration of the lumen.

Various proliferative and indurative changes in the placenta may occur as the result of syphilitic infection.

DEGENERATIONS.

Fatty and amyloid degeneration and calcification of the placental tissue are of not infrequent occurrence.

Cysts of the placenta are of occasional occurrence; their origin is in most cases obscure.[1]

THE MAMMA.

MALFORMATIONS.

Absence of both mammæ is only found in connection with other marked malformations.

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[1] See *Ahlfeldt*, Arch. für Gynäkologie, Bd. 2, p. 397. *Fenomenodes*, ibid., Bd. 15, p. 343.

Absence of one mamma has been observed in a few cases, with and without defective development of the corresponding half of the thorax.

Absence of one or of both nipples is more common.

Arrest of development of the mammæ is found in connection with arrest of development of the organs of generation, and, to a less degree, alone.

Supernumerary mammæ and nipples have been observed in a number of cases. The glands may all secrete milk during lactation.

Too early development of the mammæ is sometimes found in young children in connection with abnormal development of the organs of generation.

HÆMORRHAGE.

In young women who suffer from amenorrhœa or dysmenorrhœa, small hæmorrhages sometimes occur in the mammæ at the time of menstruation. The blood may find its way into the milk ducts and exude in small quantities at the nipple.

Contusions of the breast may produce extravasations of blood in the mammary gland or the surrounding connective tissue. This may become absorbed, or may remain and be surrounded by fibrous tissue or be converted into cysts.

INFLAMMATION.

During lactation, the nipple is liable to become inflamed in three ways, which may occur separately or be combined together.

1. The epidermis is rubbed off by nursing, the cutis becomes inflamed and converted into granulation tissue; in this way small or large ulcers may be formed.

2. Fissures are formed at the base of the nipple, which extend completely through the skin, and are lined at the bottom with granulation tissue.

3. There is a diffuse inflammation of the whole nipple, which does not, however, go on to suppuration. The nipple is conical, red, swollen, and very painful.

Acute Inflammation of the Mamma (Mastitis) occurs most frequently during lactation; it also occurs during pregnancy, and occasionally in women who are neither pregnant nor nursing.

The inflammation may involve the subcutaneous connective tissue, the gland itself, or the connective tissue between the gland and the wall of the thorax. The inflamed tissues are at first congested, swollen, hard, and painful. The inflammation may stop at this point and resolution take place; but more frequently it is succeeded by suppuration. If the inflammation involves the subcutaneous connective tissue, the abscess may be superficial and soon open through the skin. If the gland is involved, one lobule after another may become inflamed (Fig. 194), so th successive abscesses are formed in different parts of the gland. If tl connective tissue beneath the gland is inflamed, a deep abscess of lar size may be formed, which usually perforates through the skin, bı sometimes into the pleural cavity. In both these latter forms of absce there is apt to be necrosis of large portions of tissue. These abscess may cicatrize, or they may pass into a chronic condition and remain suppurating, fistulous tracts for a long time.

In new-born children, there is often a painful swelling of the breast which usually subsides in a few days, but may go on to suppuration.

Epidemic parotitis is sometimes complicated by mastitis.

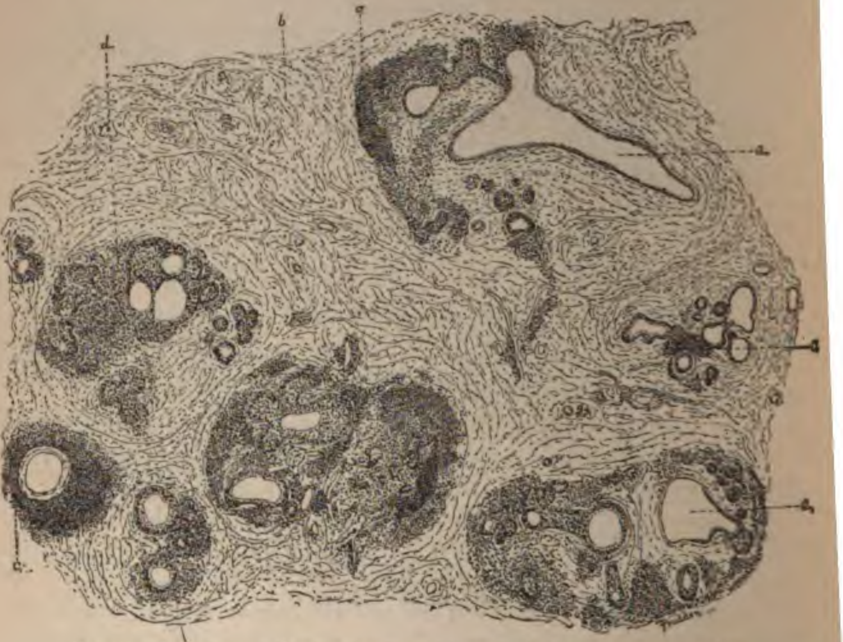

FIG 194.—SUPPURATIVE MASTITIS OCCURRING IN THE NON-FUNCTIONATING GLAND.

a, milk ducts; *b*, interstitial tissue; *c*, dense collections of pus; *d*, diffuse infiltration of lobule with pus.

Chronic Inflammation of the interstitial connective tissue of the mammary gland may result in the formation of dense connective tissue (Fig. 195), with or without cystic dilatation of the milk ducts and atrophy of the glandular elements. Acute exudative inflammation may occur in a gland which is the seat of chronic inflammation, and abscesses may be formed.

Eczema sometimes affects the skin of the nipple. Attention has lately been drawn to the relationship between this inflammation and carcinoma of the nipple, for the two are frequently associated. It is possible that the eczema may lead to the subsequent development of the carcinoma.

Syphilitic ulcers may occur in the nipple either as primary chancres or as mucous patches. Gummy tumors have been observed in the mamma.

TUMORS.

There may be a general hypertrophy of one or both breasts. This is usually found in young, unmarried women, but sometimes in advanced life. There is an increase in all the elements of the gland, both the glandular and the connective tissue.

Cysts of the mamma seem to be for the most part retention cysts, formed by the dilatation of the glandular ducts or acini. During lactation, such retention cysts are sometimes formed, and then contain milk. They may reach an enormous size. At other times, retention cysts are formed containing serous or viscid brownish fluid, which often exudes through the nipple. These cysts may be large or small, single or multiple. There is usually at the same time some growth and induration of the connective tissue of the gland. In some cases there are polypoid outgrowths of connective tissue from the wall of the cyst. These cysts are not to be confounded with the cysts which are developed with the intracanalicular tumors, of which we shall speak below.

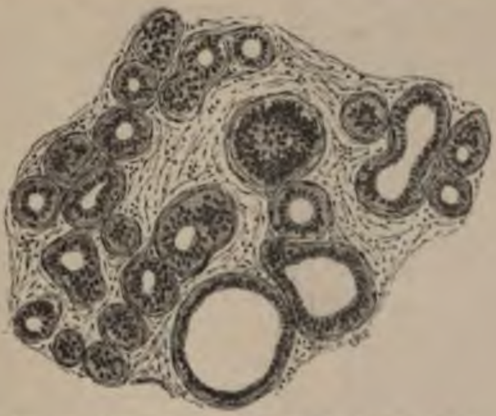

FIG. 195.—CHRONIC INFLAMMATION OF MAMMARY GLAND.

Fibroma.—Circumscribed tumors composed of connective tissue are sometimes found in the breast. They are dense and hard, and may inclose in them some of the gland ducts and acini.

Intracanalicular Fibroma.—These tumors are formed by a diffuse growth of connective tissue, a dilatation of the milk ducts, and a growth of polypoid fibrous tumors from the walls of the ducts into their cavi-

ties. The glandular acini may be atrophied, or enlarged, or cystic.
section of such a tumor looks like a solid mass of fibrous tissue, divid
by clefts and fissures lined with cylindrical or cuboidal epithelium (F
196), or containing cysts into which project polypoid fibrous outgrowt
These tumors grow slowly, but if left to themselves may reach an en
mous size. The skin over them may ulcerate and the tumor proj
through the opening in fungous masses.

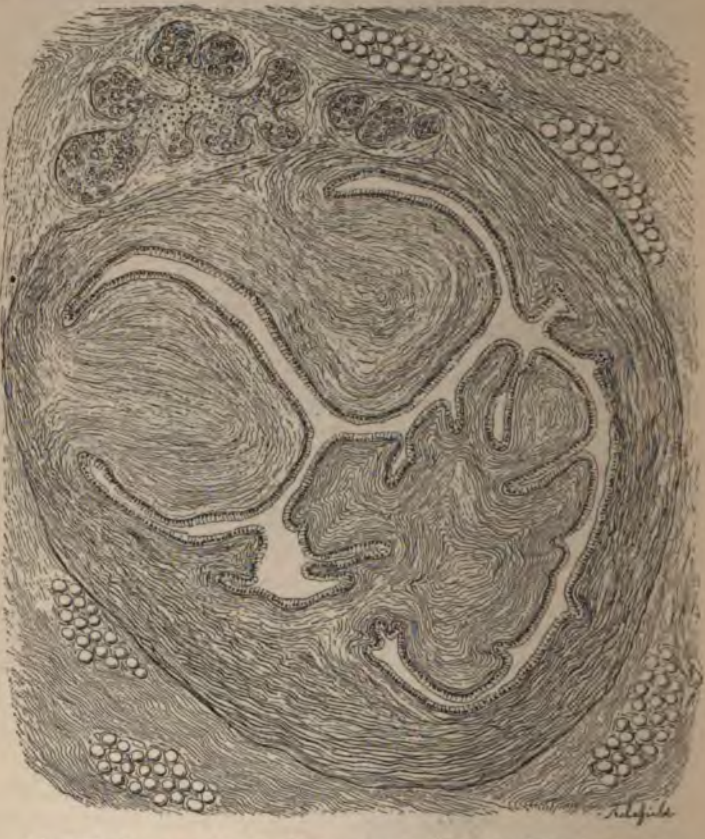

FIG. 196.—INTRACANALICULAR FIBROMA OF THE MAMMA, × 170 and reduced.
Cross-section of a milk duct with polypoid ingrowths.

Sometimes the new connective tissue forms a more or less thi
cylindrical investment of the duct without growing into its lum
This formation, which is shown in Fig. 197, is sometimes called *pe
canalicular fibroma*.

Myxoma.—This form of tumor may occur as a circumscribed grow
replacing part of the mamma; or it may be developed in the same w
as the intracanalicular fibromata. It is not uncommon in these int

canalicular tumors to find a combination of fibrous, mucous, and sarcomatous tissue in the same tumor.

Chondroma is a very rare form of tumor in the mamma. A few cases have been described in which it was combined with carcinoma.

Adenoma.—Tumors composed of glandular acini, and ducts surrounded by connective tissue, are of frequent occurrence in the mamma (Fig. 68, page 148). They are either single or multiple, or several may be developed successively in the same breast. They grow at first slowly, afterwards more rapidly. Their structure may be further complicated by the dilatation of one or more of the ducts which compose the tumor into cysts, and the ingrowth of connective tissue from the walls of these cysts.

Sarcoma.—This form of growth may be developed as a circumscribed tumor of small or large size. Its basement substance is that of connective or of mucous tissue, and may be scanty or abundant. The cells are spheroidal, fusiform, branched, or polygonal. These tumors may simply replace the gland; or glandular acini and ducts may be inclosed within them; or these ducts and acini may be dilated so as to form cysts; or there may be a new growth of the gland tissue so as to form an adeno-sarcoma.

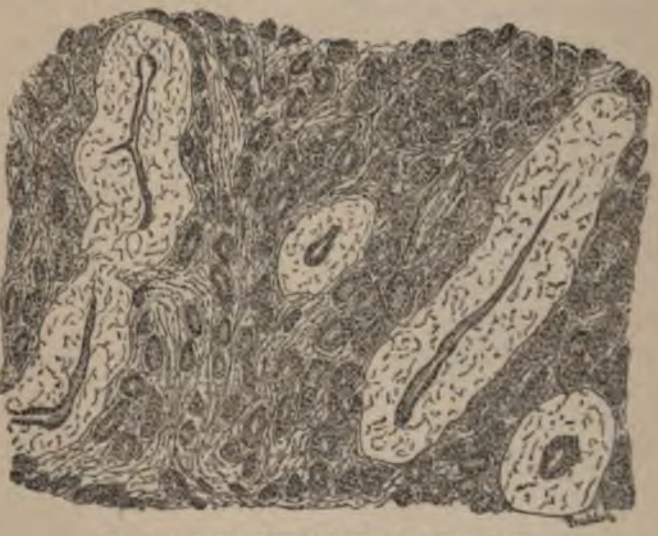
FIG. 197.—PERICANALICULAR FIBROMA OF THE MAMMA.

In other cases the sarcoma takes the intracanalicular form. There is a diffuse growth of sarcomatous tissue, a dilatation of milk ducts, and an outgrowth of sarcomatous tissue from the walls of the dilated ducts into their cavities. These tumors often reach an enormous size, and there is apt to be ulceration of the skin over them.

Carcinoma of the mamma is most common in women between the ages of thirty-five and fifty-five, but it sometimes occurs in women n over twenty years old, and sometimes in old persons. It occurs in eith breast, in the right rather more frequently than in the left, but som times in both. The growth begins more frequently at the periphery the gland than at its centre, and more frequently in the upper edge the gland than in any other place.

The growth most frequently begins as a small, circumscribed nodul which enlarges and involves more and more of the breast; sometimes, hov ever, it is diffuse from the first, and sometimes it begins in the nipple.

It may infiltrate the adjacent tissues and the axillary and cervic glands, and form metastatic tumors in different parts of the body.

There are several different anatomical forms of the growth:

1. Those in which the epithelial elements preponderate, the soft o so-called *medullary carcinomata.*

2. Those in which both the connective-tissue stroma and the epithelial cells are both prominently developed, the cells lying in well defined larger and smaller irregular-shaped spaces, so that the simulatio of gland tissue is tolerably close. These are called *carcinoma simplex.*

3. The tumors in which the connective-tissue stroma preponderates, giving the tumor its hard, dense character. This is the *carcinoma fibrosum,* or *scirrhous.*

Colloid carcinoma of the mamma is rare. Various secondary changes may occur in these tumors, such as have been described in the section on Tumors.

In any of these forms of cancer there may be cystic dilatations of the ducts and acini.

Besides the primary cancers of the mamma, secondary cancers are met with in rare cases.

MALE.

THE PENIS.

MALFORMATIONS.

Entire absence of the penis is met with in connection with great defects of development of the rest of the body.

Absence of the penis, with proper development of the other organs of generation, is rare. The urethra then usually opens into the rectum.

An abnormally small penis is found with absence or arrested development of the testicles.

Absence or a rudimentary form of the prepuce has been observed in a number of cases. Congenital phimosis is also not infrequent.

Hypospadia consists in an arrest of development of the penis and scrotum. In its highest degrees, the penis is one-half to one inch long, the glans penis small and resembling a clitoris. On the lower side of the penis is a deep cleft lined with mucous membrane. Into this cleft the urethra opens at the root of the penis. The scrotum remains separated into two halves, resembling labia majora. The testes descend into their proper position on each side or remain in the abdomen. If the testicles continue to develop normally, the individual has the appearance and capacities of a man; if their development is arrested, the individual is small and has a womanish appearance.

In lesser grades of the same malformation, the two halves of the scrotum are joined and the penis is larger, but a part of the urethra remains open as a cleft at any point of the penis.

Epispadia is an opening of the urethra on the upper side of the penis. It presents various grades and forms.

Hermaphroditism.—This is a union of two sexes in the same person, the test of which is the presence of the secreting organs, the ovaries, and testicles. True hermaphroditism is rare, but it does occur, while most of the conditions called hermaphroditism are in reality due to varying malformations of the external generative organs.

Pseudo-hermaphroditism.—This malformation consists in an abnormal change in the transition from the fœtal condition of the parts to their fully developed form. In the male, normally, the greater part of Müller's canal disappears, and its lower end forms the vesicula prostatica. In this malformation, Müller's canal is changed, as it is in the female, into Fallopian tubes, uterus, and vagina, while at the same time the testes, epididymes, vesiculæ seminales, and spermatic cord are formed as usual. In the lesser degrees of this malformation, we find, in the place of the vesicula prostatica, a pear-shaped sac as large as a pigeon's egg, with muscular walls and an epithelial lining. This sac may be incompletely divided into a uterus and vagina, and it opens into the urethra. In the higher grades, we find a well-formed vagina and uterus. The uterus may or may not have Fallopian tubes. The testicles are usually retained in the abdomen or inguinal canals, and are small. The spermatic ducts run on the sides of the uterus and open into the urethra or are closed. The penis and scrotum appear as in hypospadia, or are well formed. The appearance of the individual varies with the development of the testicles.

True Hermaphroditism may be lateral. In this condition there is hypospadia; a vagina and uterus and a Fallopian tube and ovary on one side, and a testicle and spermatic cord on the other.

In certain cases, which may be called *bilateral hermaphroditism*, there is a testicle on one side and an ovary on the other.[1]

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[1] For a detailed consideration of the malformations of the male and female generative organs, consult *Klebs*, "Handbuch der pathologischen Anatomie," and more recent cases of hermaphroditism by *Heppner*, Arch. f. Anat. u. Physiol., 1870, and by *Hofmann*, Wien. Med. Jahrb., 1877.

Enlargement of the penis is sometimes caused by venous cong from heart disease; by long-continued masturbation, as a result of the corpus cavernosum may lose its contractility; and in rare ca hypertrophy of the stroma of the corpus cavernosum.

Injury and Hæmorrhage.—Injuries to the penis are liable tc rise to severe hæmorrhage on account of its peculiar vascular char suppurative inflammation, gangrene, infiltration with urine and it sequences, are also liable to occur. The contractions of the cica tissue by which wounds are healed frequently give rise to variou tortions of the organ and not infrequently prevent subsequent erec

INFLAMMATION.

Balanitis, inflammation of the prepuce, is usually produced by g rhœa or by accumulations of smegma. The skin is red and sv and may ulcerate. Condylomata may be formed, and adhesions be the prepuce and glans. The glans may ulcerate and the prepuce n much thickened. If the prepuce is long, there is an inflamn phimosis, and the products of inflammation accumulate within the len prepuce. In some cases, the prepuce becomes gangrenous.

Paraphimosis is produced by the retraction of a narrow prepu hind the glans, with consequent stricture, inflammation, and som gangrene.

Inflammation of the Corpora Cavernosa is produced by gonor injuries, fistulæ, typhus fever, measles, and occurs in connectio inflammation of the connective tissue of the pelvis. It results in f induration of portions of the corpora cavernosa; in rare cases, scesses or diffuse purulent infiltration; sometimes in gangrene.

Syphilitic Ulcers frequently occur on the glans penis and pr The indurated chancre is formed either from an excoriation in w pustule is formed or from a little nodule. The pustule breaks a walls are infiltrated with small round cells. The nodule softens, down, and forms an ulcer, of which the walls are infiltrated with c the same way.

Syphilitic condylomata are of frequent occurrence on the glans

Phagedænic ulcers occur and may destroy a considerable part penis. Herpes of the prepuce occurs in the form of small vesicles, may later become ulcers. Erysipelatous and furuncular inflamn sometimes involve the skin of the penis.

TUMORS.

Papilloma is found on the prepuce and glans penis. It occurs form of little warty growths, or of composite, cauliflower masses as large as a fist. In either case, the structure is the same, hypertr papillæ covered with epithelium. Sometimes the epithelial layers become thick and horny, forming large, dense projections.

Fibroma diffusum, or elephantiasis of the prepuce, may occur, leading to immense thickening of the structure. It consists in a diffuse growth of the deep fibrous tissue of the cutis. *Lipomata, angiomata, circumscribed fibromata*, and sebaceous cysts may occur in the penis. *Carcinoma* of the penis usually occurs in the form of epitheliomata. These are most frequent in the prepuce and glans penis. They may have the form of flat ulcers, or of infiltrating, ulcerating nodules, or very frequently assume the form of papillary outgrowths, which may attain great size, ulcerate, or undergo a variety of inflammatory changes. These growths may involve the entire skin of the penis; they may invade deeper parts. The inguinal glands may be invaded. Distant metastases may occur, but are not frequent.

Glandular carcinoma of the penis is not common. It may be secondary to carcinoma in some other part of the body.

Calcification and *Ossification* of the connective tissue of the corpora cavernosa sometimes occur. Large and small preputial calculi are occasionally found between the prepuce and the glans. These may be formed in situ, may come from the bladder or from without, and may later increase in size.

THE SCROTUM.

The skin of the scrotum is subject to the various forms of lesions which may occur in any part of the integument.

Elephantiasis of the scrotum consists in the main of a development of new connective tissue from the cutis, which is sometimes accompanied by dilatation of the lymph vessels. The thickened scrotum sometimes forms very large tumors, often rough upon the surface, which may entirely cover in the penis. *Lipomata, fibromata, atheromatous* or *sebaceous cysts*, and *dermoid cysts* containing hair, bone, cartilage, etc., are sometimes found. Occasionally the skin of the scrotum is beset with numerous larger and smaller sebaceous cysts, which raise the surface into little wart-like projections. *Epitheliomata*, in the form of flat or papillary ulcerating tumors, are of frequent occurrence among chimney sweepers, and may lead to extensive ulcerations of the adjacent parts and involvement of neighboring lymph nodes.

In very rare cases, tumors containing a considerable portion of a fœtal skeleton have been found in the scrotum.

THE TESTICLES.

MALFORMATIONS.

Absence of both testicles, either with or without absence of the e didymes, spermatic cords, and vesiculæ seminales, occurs in rare case The scrotum is only indicated or may contain the epididymes. T penis is small, and the individuals are small and poorly developed.

Instead of being entirely wanting, the testes may be imperfect developed. The individuals are weakly and effeminate. Absence of o testicle, with healthy development of the other, is more frequent. T corresponding epididymis and cord may be absent or present.

The spermatic cords and vesiculæ seminales may be absent or impe fectly developed on one or both sides, while the testes are normal.

Either one or both testicles may remain permanently in their fœt position, or may not descend into the scrotum for several years aft birth (*cryptorchismus*). Their descent may even be delayed until th thirtieth year of life. This condition may depend on an arrest of d velopment in the testes or the gubernaculum testis; on adhesions pr duced by intra-uterine peritonitis; on narrowing of the inguinal canal on narrowing or shortening of the vaginal process of the peritoneum; o on abnormal size or position of the testicle. Usually the malformatio is confined to one testicle, and then is more frequent on the left side The testicle is usually found in the abdomen close to the mouth of th inguinal canal, or in the inguinal canal just below the external ring; but it may be beneath the skin in the perineum, or in the crural canal with the femoral vessels, or elsewhere. The retained testis is usually not fully developed, or undergoes fatty or fibrous degeneration. The retention of one or even of both testicles does not preclude the possibility of procreation. Retained testicles are prone to inflammatory changes and liable to become the seat of malignant tumors.

Sometimes, while the testis is retained, the epididymis and spermatic cord descend into the scrotum. In rare cases, the position of the testis may be changed, so that the epididymis and cord are in front. The existence of a supernumerary testis has been asserted in some cases, but is rather doubtful.

Atrophy of the testicle may occur in old age or in persons who are in a condition of premature senility; or as the result of pressure from herniæ, hydrocele, or inflammatory products.

HYDROCELE.

Hydrocele of the tunica vaginalis consists in an accumulation of serum in the cavity of this membrane. It is usually confined to one side. It is caused by acute or chronic inflammation of the tunica vaginalis, by varicocele, or by general dropsy. The serum is found in small or in large quantities; it is usually transparent, may contain cholestearin, or be purulent and contain the pyogenic bacteria, or be mixed with blood. The tunica vaginalis remains unchanged, or is thickened, or contains plates of bone, or is covered with polypoid fibrous bodies, which fall off and are found free in the cavity of the sac. There may be adhesions between the layers of the tunica vaginalis, and in this way the fluid becomes sacculated. The testis is pushed downward and backward; it remains unchanged or is atrophied.

Hydrocele of the processus vaginalis consists in an accumulation of serum in the cavity of the vaginal process of the peritoneum, which remains open after the descent of the testicle. There are several different varieties.

(*a*) The vaginal process is entirely open, and there is a free communication with the peritoneal cavity. The serum may originate in the cavity of the peritoneum or of the vaginal process, and passes freely from one to the other.

(*b*) The processus vaginalis is closed in the inguinal canal while its lower portion is filled with serum.

(*c*) The processus vaginalis is closed about the testis, and the visceral layer of the tunica vaginalis is formed. The serum accumulates in the upper part of the vaginal process, which communicates with the peritoneal cavity.

(*d*) The vaginal process is closed in the inguinal canal and over the testis; the serum accumulates so as to form one or more sacs between these two points. Inguinal hernia may complicate this form of hydrocele.

Hydrocele of the spermatic cord consists in a general œdema of the connective tissue of the cord, or in the development of circumscribed cysts in this connective tissue.

A peculiar form of hydrocele is produced by the accumulation of serum in the sac of an inguinal hernia, from which the intestine has become retracted.

SPERMATOCELE.

Cysts containing spermatic fluid not infrequently arise from the epididymis or from the rete testis. These sometimes acquire a large size and crowd the tunica vaginalis before them, so that they simulate a collection of fluid in the cavity of the latter. The wall of the cyst may be lined with ciliated or with flattened epithelium. The contents are sometimes simply serous, but more frequently opalescent and contain great numbers of spermatozoa.

HÆMATOCELE.

Hæmatocele of the tunica vaginalis consists in an effusion of blood into the cavity of this sac. It may be produced by injury; in scurvy, or the hæmorrhagic diathesis; or it may complicate a pre-existing hydrocel The effused blood usually soon degenerates, and we find the sac fill with a brownish fluid or a thick, grumous mass. The tunica vaginal may be thickened. The testis remains normal or is atrophied.

Effusion of blood into the loose connective tissue of the scrotum often called *extravaginal hæmatocele.*

Hæmatocele of the spermatic cord occurs in rare cases as a diffus infiltration of blood in the connective tissue of the cord. Or blood ma be effused into a hydrocele of the cord.

INFLAMMATION.

Inflammation of the testicles may be caused by injuries, exposure t cold, inflammation of the urethra, syphilis; or it may occur in parotiti The testes, epididymis, or tunica albuginea may be principally involved Usually only one testicle is inflamed, sometimes both. The inflammation may extend to the vas deferens. The inflammation may be acut or chronic.

Acute Orchitis is most frequent in the epididymis and tunica albuginea. When the testis is involved, the organ is congested and infiltrate with serum or pus. From this condition it may return to the norma state, or small abscesses may form which may be absorbed, or the may increase in size so as to involve nearly the entire organ. They may perforate externally, and then healing may occur by means of granulation tissue; or extensive gangrenous destruction of the scrotum may occur. They may become inclosed in a fibrous capsule, and the contents dry and become cheesy or calcified, and so persist for a long time. The acute inflammation may pass over into the chronic form. *Acute epididymitis* is frequently the result of gonorrhœal infection, and may or may not be associated with inflammation of the testis.

The products of inflammation may collect in varying quantity in the lumina of the seminiferous tubules, and in the ducts of the epididymis, and the epithelium of these structures may degenerate.

Chronic Orchitis occurs as a sequel of acute inflammation or as an original condition. It may affect the testis, the epididymis, or the spermatic cord. The seminiferous tubules may be filled with desquamated and degenerated epithelium; they may be atrophied, or their walls may be greatly thickened so that they are converted into dense fibrous cords, with almost or quite complete obliteration of their lumina. There is usually a marked increase in the interstitial tissue, which causes atrophy of the tubules (Fig. 198). The albuginea may be greatly thickened. In some cases, the testis is converted into a mass of dense connective tissue, in which but little trace of the original structure can be made out. The new-formed connective tissue may become calcified. A periorchitis may lead to thickening and union of the layers of the tunica vaginalis testis. Abscesses are not infrequent in connection with chronic orchitis.

Tubercular Orchitis may occur in connection with tuberculosis of the other genito-urinary organs or the lungs, in acute general miliary tuberculosis, or by itself. It usually begins in the epididymis, and may extend from there to the testis; or it may commence in the testis itself. The appearances which the testicles present when the seat of this form of inflammation are exceedingly varied and difficult of interpretation. This is partly due to the complex structure of the organ, partly to the varied complicating simple inflammatory changes which the different parts of the organ undergo in connection with the special tubercular inflammation, and the impossibility of making any definite morphological distinction between them. Further researches are urgently needed in this direction, and it seems probable that in the presence or absence of the tubercle bacillus we shall find the needed differentiating factor between various inflammatory processes which are at present grouped under the general heading of tuberculosis testis.

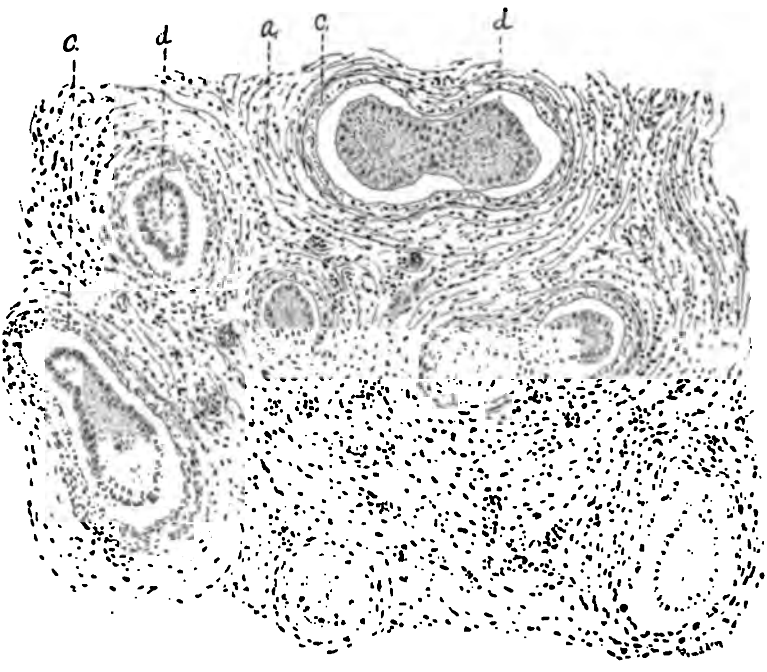

FIG. 198.—CHRONIC INTERSTITIAL ORCHITIS WITH ATROPHY OF THE SEMINIFEROUS TUBULES.
a, thickened interstitial tissue; *c*, thickened membrana propria of the tubules; *d*, separated epithelial cell mass in the lumen of the tubules.

We may find in the testicle small circumscribed masses of cells, visible to the naked eye as whitish spots, which are sometimes composed small spheroidal cells or of larger polyhedral or fusiform or round cell These occur in the walls of seminiferous tubules and blood-vessels an in the interstitial tissue. Sometimes associated with these smalle nodules, and sometimes not, we find larger, irregular yellowish or gra cheesy masses, which are believed by many to be formed by the conflu ence and degeneration of the smaller nodules. The cheesy masses ma break down and open externally, giving rise to fistulæ, gangrenous in flammation, etc. Hand-in-hand with this nodular formation of tissue which is disposed to degenerative changes, there are various more or les diffuse alterations of the parenchyma and interstitial tissue of the orga which must not be overlooked, and which often constitute a most prom inent and important factor in the lesion. The interstitial tissue may b

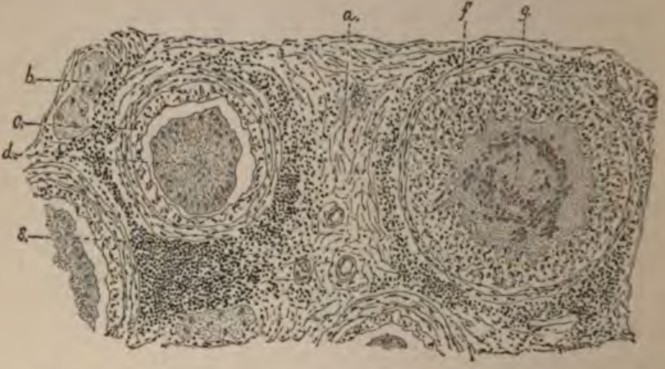

FIG. 199.—CHRONIC ORCHITIS WITH THE FORMATION OF STRUCTURES RESEMBLING MILIARY TUBERCLES. *a*, thickened interstitial tissue; *b*, mass of granular cells in the interstitial tissue; *c*, thickened membrana propria of seminiferous tubule; *d*, mass of separated epithelium in tubule; *e*, accumulation of small spheroidal cells around tubules; *f*, thickened membrana propria inclosing *g*, a multinuclear mass resembling a giant cell.

more or less densely and diffusely infiltrated with small spheroidal cells. The arteries are often the seat of obliterating endarteritis. The walls of the seminiferous tubules may be very much thickened, so that the lumen may be entirely obliterated. The epithelium lining the tubules may be fatty, disintegrated, and peeled off, or it may have largely disappeared. The lumen of the tubules may be filled with a granular, nucleated mass which in transverse sections looks like a giant cell. The thickened walls of the tubules may be infiltrated with small spheroidal cells, so that the underlying stroma is scarcely visible. When this occurs in connection with a similar infiltration of the interstitial tissue and the formation of giant cells in the lumina, we have structures which present the greatest resemblance to some forms of tubercle granula (Fig. 199).

Tubercular inflammation may extend from the testis to the vas deferens, vesiculæ seminales, and prostate.[1]

Syphilitic Orchitis.—This may occur in the form of a diffuse new formation of connective tissue, which may occur in some particular part of the organ or be widely distributed through it, and by reason of which the organ becomes dense and firm. Morphologically, there is no difference between this form of orchitis and chronic indurative orchitis from other causes. It may occur in children affected with congenital syphilis.

Gummata may form in connection with the interstitial induration. These may disappear, leaving irregular cicatrices.

TUMORS.

Fibroma occurs in the form of small dendritic or polypoid growths of the visceral layer of the tunica vaginalis. These sometimes become free, and are found in the sac, usually in connection with hydrocele. Small nodular fibromata occasionally occur in the albuginea and in the spermatic cord.

Lipomata, either pure or in combination with myxoma and sarcoma, may arise from the connective tissue of the spermatic cord, or from the tunica albuginea.

Chondroma, sometimes in a pure form, but more frequently combined with myxoma and sarcoma, occur in the testicles, and may attain a large size. *Osteoma* has been described.

Sarcomata occur in the testes and epididymis, most frequently in the former. They present the greatest variety in structure. They may be composed of spheroidal or spindle-shaped cells; they may be soft or contain much fibrous tissue; they are very frequently combined with myxoma, chondroma, lipoma, etc. Owing to the occlusion of the seminiferous tubules, cysts may be formed in these sarcomata. In these cysts polypoid growths of sarcomatous tissue may occur in the form of intracanalicular growths. Thus the so-called *cysto-sarcomata* of the testicle are formed. The walls of these cysts may coalesce, so that large, irregular cavities may be formed. When the cysts are not filled by polypoid outgrowths from their walls, they may contain a mucous, serous, or bloody fluid, or masses of flattened cells, fat, and cholestearin. The cysts may be lined with cylindrical, ciliated, or flattened cells.

Rhabdomyomata have been several times observed, frequently in combination with cysts.

Adenoma is occasionally found, usually in combination with sarcoma, or carcinoma, or with cyst formation.

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[1] For the literature of tuberculosis of the testicle consult *Waldstein*, "Zur Kenntniss der tuberculösen Erkrankungen des Hodens." Virch. Arch., Bd 85, p. 377, 1881.

Carcinoma of the testicle is commonly of the soft medullary form, of rapid growth, and usually primary. It may commence in the testis or epididymis. Usually only one testicle is involved. Frequently the entire glandular portion of the organs is replaced by the new growth. The albuginea expands with the growth of the tumor, and may continue to inclose it even when of large size. The tissues are often very vascular, and hæmorrhages, areas of softening, fatty and mucous degeneration are frequent. The inguinal and lumbar lymph nodes are apt to become involved, and distant metastasis may occur. Rarely the growth assumes a scirrhous form.

Cysts.—Aside from the above-mentioned cysts which occur in connection with tumors and spermatocele, cysts may be formed from persistent remnants of Müller's canal in the epididymis, or from obstruction of the seminiferous tubules or ducts by inflammatory products or tissue.

Dermoid Cysts of various kinds are of infrequent occurrence, and are sometimes quite complex in character. They may be imbedded in the substance of the gland. Probably some of the above-mentioned cystic rhabdomyomata belong here.

PARASITES.

Echinococcus may occur in the testis or epididymis.

THE SEMINAL VESICLES.

The seminal vesicles may be the seat of acute or chronic inflammation, which is most frequently connected with inflammatory changes in adjacent parts, prostate, urethra, etc. As a result of chronic inflammation, the vesicles may be atrophied, or they may be greatly dilated as a result of constriction of the ducts.

Tubercular inflammation is usually secondary. Carcinoma of the rectum or other genito-urinary organs may secondarily involve the seminal vesicles. Small concretions, sometimes containing masses of spermatozoa, are occasionally found in the seminal vesicles.

THE PROSTATE.

Hypertrophy of the prostate is a frequent senile change; it is general or partial.

In general hypertrophy, the entire organ is enlarged, and may reach the size of a man's fist. The enlargement is symmetrical, or is most marked in one half or in the so-called middle lobe. The organ is hard and dense, or soft or alveolar, containing numerous small openings from which a turbid fluid exudes. These different appearances depend upon the character of the hypertrophy. The muscular and fibrous tissue alone may be increased, which is most common, or at the same time the glandular tissue, or the glandular tissue alone. In the latter case, the lesion is more properly an *adenoma*. The increase of muscular tissue properly constitutes a *myoma*.

In partial hypertrophy, we find circumscribed nodules of muscular tissue or of muscular and glandular tissue. They are usually situated at the periphery of the organ and project into the bladder. They may become detached from the prostate, and are found as small, movable tumors beneath the mucous membrane of the bladder.

Both forms of hypertrophy frequently produce, by pressure, retention of urine and changes in the bladder.

Atrophy of the prostate is sometimes seen in connection with atrophy of the testicles. with castration, and as a result of inflammation. Sometimes the ducts of the glandular portion are enlarged, or there may be fibrous degeneration of the organ.

INFLAMMATION.

Inflammation of the prostate is caused by gonorrhœa, by injuries, or, more rarely, is idiopathic. It may run an acute or chronic course. The gland may after a time return to its normal condition, or is gradually converted into a mass of fibrous tissue filled with abscesses. The abscesses may perforate into the bladder, urethra, vesiculæ seminales, rectum, or peritoneum. Or the inflammation may extend to the connective tissue of the scrotum or beneath the pelvic peritoneum. The pus may become thickened and cheesy, or even calcified.

Tubercular Inflammation of the prostate usually accompanies a similar lesion of some of the other genito-urinary organs, and is rarely of primary occurrence. Large cheesy masses are often formed, which may break down and open into the bladder or rectum.

TUMORS.

Adenoma of the prostate occurs in one of the forms of hypertrophy of the gland, either with or without an increase in the fibro-muscular interstitial tissue.

Carcinoma is of occasional occurrence, and may be primary or secondary.

Cysts of the prostate are sometimes found either as a result of occlusion of the ducts by hypertrophy of the interstitial tissue, tumors, etc., or as a result of faulty development.

PARASITES.

Echinococcus of the prostate has been described, but is rare.

CONCRETIONS.

Small ovoidal or spheroidal bodies having the characters of corpora amylacea are of very frequent occurrence in the alveoli of the prostate, particularly in old persons. We find a certain number of them in the prostate of nearly all old men, but they are sometimes present in great numbers. Larger, irregular concretions, apparently formed by the coalescence or growth of the smaller ones, are less frequently found, and may be incrusted with lime salts. These concretions may give rise to ulceration of the ducts of the gland or to interference with the passage of urine, but in a majority of cases they seem to be of little or no practical importance.

THE MALE MAMMA.

There may be an abnormal number of mammæ. In boys, at about the time of puberty, the mammæ may be swollen and inflamed, or they may secrete milk Cases are recorded in which adult males possessed large mammæ which secreted milk. The breasts may be enlarged from an increase of fat or of connective tissue.

Cysts of the male breast are not very infrequent. *Fibromata, sarcomata, cysto-sarcomata, myxomata,* and various forms of *carcinomata* are recorded.[1]

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[1] For literature of tumor of male mamma, see *Gross*, "Tumors of the Mammary Gland," p. 237.

THE BONES.

DISTURBANCES OF CIRCULATION.

Hyperæmia.—The evidences of this condition are most marked to the naked eye in the periosteum and marrow, particularly the latter. It should be remembered that the color of the marrow varies considerably under normal conditions, depending upon age and situation. In the bones of the fœtus and new-born, and near the areas of ossification in the young, the marrow is normally red in color. In adults, the marrow of the sternum, vertebræ, and to a certain degree that of the ribs, pelvic and cranial bones, and the cancellous tissue of the ends of the long bones, is red or reddish in color. But most of the marrow, particularly in long bones of the extremities, is of a yellowish color from the presence of fat cells. In old age the marrow of all the bones is apt to become pale, and to assume a more or less translucent or gelatinous appearance.

Hyperæmia usually occurs as an accompaniment of inflammatory processes in the bone, and, when marked, the periosteum is swollen and red; the compact bone tissue may appear of a pink color, while the marrow, either by an increase in the amount of blood or absorption of its fat, or both, may be of a uniform dark-red color, or mottled with red and reddish yellow.

Hæmorrhage.—This may be due to wounds and injuries, to inflammatory and necrotic processes; and small hæmorrhages often accompany scurvy, purpura, hæmorrhagic diathesis, and leukæmia. Hæmorrhages of considerable size between the periosteum and bone may lead to serious consequences, by cutting off the blood-supply to the superficial layers of bone, and thus inducing necrosis; but when not in contact with the air, they are not usually of serious import, since they are readily absorbed. The smaller hæmorrhages of the medulla are not usually of much importance. The decomposition of the extravasated blood may lead to extensive pigmentation of the marrow.

WOUNDS, FRACTURES, AND DISLOCATIONS.

For details of the varied alterations produced under these conditions, and the secondary changes involved in the healing process, we refer to the works on surgery. It may be stated here, however, that the healin of fractures occurs by the formation of granulation tissue in greater o less amount about the seat of fracture, and the direct formation of bon under the influence of osteoblasts, or by a preliminary formation of cai tilage or fibrous tissue and the gradual conversion of this into bone b metaplasia.

INFLAMMATION.

The periosteum, bone tissue, and marrow are so intimately connecte that in most cases they all share to a greater or less degree in the patho logical alterations of the bones. But as sometimes one, sometime another is most markedly involved, it is convenient to consider separately here the inflammatory changes by which they are respectively affected.

Periostitis.

We may distinguish several varieties:

1. *Simple Acute Periostitis.*—This form is apt to occur in children and ill-nourished persons from comparatively slight injuries or from unknown causes. The periosteum is thickened, succulent, congested, and more or less abundantly infiltrated with leucocytes, while the connective-tissue fibres are swollen. The periosteum becomes less firmly adherent to the bone, and the cells of the inner layers are increased in number. This variety of inflammation may terminate in the disappearance of the new elements and complete resolution; or it may represent a preliminary stage of one of the other varieties of inflammation.

2. *Suppurative Periostitis* may begin as a simple or as a purulent inflammation. The pus is formed in the inner layers of the periosteum, and between it and the bone. The outer layers of the periosteum may resist for a long time the suppurative process. The accumulation of pus may dissect up the membrane from the bone and leave the latter bare. The pus thus formed may remain in this position for a long time, may be absorbed, may become dry and cheesy, or may burst through the periosteum and form abscesses in the soft parts. The bone, if separated from its nutrient membrane, may remain unchanged, but more frequently necrosis, or inflammation of the bone itself, is set up. Such a periostitis may run an acute or a chronic course.

Sometimes suppurative periostitis takes on a very *malignant* character. Pus is developed not only beneath but in the periosteum, forming abscesses filled with foul pus. The periosteum breaks down into a gangrenous foul-smelling mass, and the same change may affect the neighboring soft parts. The medulla may take part in the process and break down into a purulent, gangrenous mass. Hæmorrhages may complicate the process. The lymphatic nodes are enlarged and swollen; abscesses may form in different parts of the body, and the patient may die with the symptoms of septicæmia. The pyogenic cocci may be found, under these conditions, in the exudations of the periosteum as well as in the metastatic abscesses.

3. *Fibrous Periostitis.*—This is a low, chronic form of inflammation, resulting in the formation of new connective tissue in the periosteum, which becomes thickened and dense and unusually adherent to the bone. It may be the result of necrosis, chronic arthritis, chronic ulcers of adjacent soft parts, etc. It may follow a simple acute periostitis.

4. *Ossifying Periostitis* results in the formation of new bone from the inner layers of the periosteum. The masses of new-formed bone, called *osteophytes*, are of variable shape. They may form a thin, velvet-like, villous layer; or they are little spiculæ; or they form larger, rounded masses, or a thick, uniform layer, extending over a large part of a bone. They may be at first very loosely connected with the bone. The new bone has at first a loose, spongy character. It is formed of thin plates of bone inclosing large cavities filled with marrow. Layers of compact bone tissue are formed from the medulla on the sides of the original plates, and thus the medullary cavities are gradually filled up with bone. The new bone may thus become as compact or even denser than normal bone. The hyperostoses and exostoses thus formed may remain indefinitely, or they may gradually become smaller and finally disappear by absorption.

The formation of new bone in the form of osteophytes, or in dense masses beneath and in the periosteum, occurs as a result of the same process by which bone tissue is normally formed. Certain rather large cells, called *osteoblasts*, which are formed along the blood-vessels, possess the power of depositing osseous basement substance about themselves, and so forming bone. Pathological new formation of bone differs from the normal mainly in the conditions under which it occurs. The blood-vessels around which the pathological bone develops, which grow out of the old vessels, as in the formation of granulation tissue, are irregularly arranged and subject to a variety of abnormal nutritive and mechanical conditions, so that the new bone is not usually formed in a series of definite systems of lamellæ, but, as above described, in a series of irregular spiculæ or masses. Moreover, as will be seen further on, the conditions under which it is formed being liable to change, and itself serving no definite purpose in the economy, as does normal bone, pathological new bone is often an evanescent structure. The details of its disappearance will be considered below.

5. *Syphilitic Periostitis.*—Syphilitic poisoning may give rise to simple, purulent, fibrous, and ossifying periostitis. Or, in addition to these, gummy tumors may be developed in the periosteum. The bone tissue is usually more or less involved. The gummata may be absorbed

or undergo cheesy degeneration, or be converted into fibrous tissue, they may suppurate.

6. *Tubercular Periostitis.*—In badly nourished persons, particular in children suffering from the so-called scrofulous diathesis (see pa 306), a chronic purulent periostitis is frequently associated with the fo mation of miliary tubercles. Abscesses are apt to form in and abo the periosteum, and when these are evacuated granulation tissue m develop, in which miliary tubercles are formed. In these tubercles t Bacillus tuberculosis may be found. The bone is apt to be involved to greater or less extent in the form of inflammatory changes or caries.

Osteitis.

Inflammation in bone tissue is dependent upon the same general conditions and presents essentially the same series of phenomena as inflammation in other kinds of connective tissue. But it is variously modifie in detail by the peculiar dense and unyielding character of the bas ment substance, and by certain peculiarities of the blood-supply and th nutritive conditions under which the cells are placed. In simple exudative inflammation, the same series of phenomena occur in connection wit the blood-vessels, resulting in the production of serum, fibrin, and pus as in other tissues; but the extent to which these changes can occur i limited, and constantly associated with striking alterations in the basement substance. It is these secondary alterations in the basement substance which lend to inflammations of the bone their most peculiar characters, and in the prominence which these assume the fundamental alterations are often overlooked. The most common of these secondary alterations are the absorption of the hard basement substance of the bone and its replacement by, or conversion into, young cellular forms of fibrillar connective tissue or marrow tissue, and the new formation, in more or less atypical manner, of new bone. As a result of these changes, the bones in simple inflammation undergo alterations either in the direction of greater vascularity and increase of the spaces filled with granulation or marrow tissue, and so become more porous and less compact at the expense of the dense basement substance; or they undergo alterations in the direction of an increase in density at the expense of new-formed or pre-existing marrow spaces. Or, as is frequently the case, both series of changes occur either simultaneously in different regions, or follow one another, or are variously associated together. Very frequently one or the other of the opposing forms of alteration predominate, or one may occur to the exclusion of the other, and we thus have two prominent forms of inflammation, which are called *rarefying osteitis* or *osteo-porosis*, and *condensing osteitis* or *osteo-sclerosis*. The exact nature of the conditions under which in one case the bones become more, in another less dense, we do not understand.

In addition to these phases of inflammation in bone, and in frequent and varied association with them, there are alterations leading to death and destruction of bone tissue in greater or less amount, which we call *caries* and *necrosis,* and also inflammatory changes, more or less characteristic, due to the influence of peculiar specific agencies, such as the syphilitic and tuberculous infection, and we thus recognize *tubercular* and *syphilitic osteitis.* Again, the production of pus is so prominent a feature in some cases as to represent a *purulent* phase of the inflammatory process. Finally, any of these forms, and commonly several of them at once, are variously associated with more or less marked inflammatory or degenerative alterations of the periosteum on the one hand, or the marrow tissue on the other, or of both combined.

Rarefying Osteitis consists essentially in the formation in the marrow spaces, Haversian canals, or beneath the periosteum, of new, very cellular and vascular tissue, resembling granulation or young marrow tissue, in connection with which, or under whose influence, the basement substance of the bone is absorbed. The absorption of the bone occurs chiefly in the same way in which the bone is absorbed in normal growth, namely, under the influence of certain large cells, called *osteoclasts,* which are grouped around the blood-vessels. If we examine a thin section of bone which is undergoing absorption (Fig. 200), we find the edges of the bone which border on the vascular surfaces irregularly indented by deep or shallow depressions, sometimes simple, sometimes quite complex. These are called *Howship's lacunæ* and are usually filled or lined by larger and smaller granular, frequently multinuclear cells—the so-called *osteoclasts.*

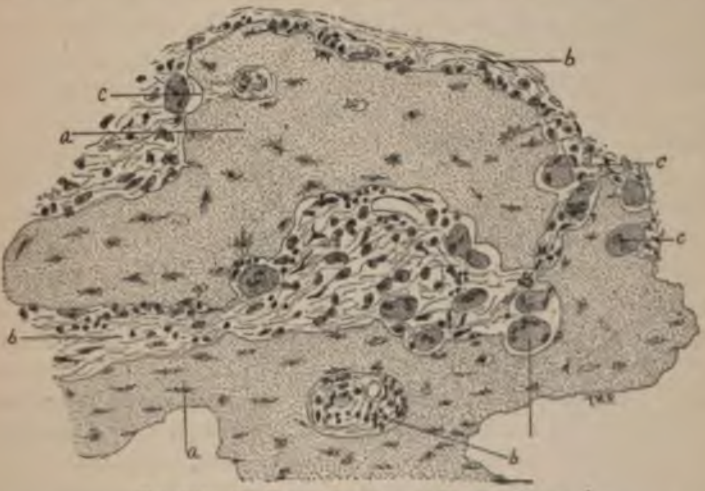

FIG. 200.—RAREFYING OSTEITIS IN ULNA OF CHILD.

a, isolated bone fragment with rough edges; *b*, marrow tissue; *c*, Howship's lacunæ with osteoclasts.

In the larger lacunæ there may be granulation tissue with loops blood-vessels, with or without cells which have the morphological ch acters of osteoclasts. Under the influence of these peculiar cells, or the new vascular tissue, the bone is gradually absorbed. In other ca we find irregular branching channels through the bone across the lam læ, which appear to be due to the enlargement and coalescence of t lacunæ and canaliculi, without the direct influence of blood-vessels other cells than the fixed cells of the bone. The tissue which repla the absorbed bone may be very rich in small spheroidal cells, or it m be more or less fibrillar. As a result of this process, irregular islets bone tissue may be entirely separated from adjacent bone and surround by a more or less fibrillar vascular tissue; this is most apt to occur in t cancellous tissue. Or the originally compact bone may become travers by a series of larger and smaller irregular branching, communicati channels with ragged walls. These progressive alterations may cea and be succeeded by a new formation of bone along the edges of t channels or cavities; it may result in necrotic changes; the vascul changes may become prominent, and suppuration ensue.

Rarefying osteitis may occur as an idiopathic disease from unknow causes; it is often associated with the scrofulous diathesis, with diseas of the joints, with fractures or other injuries to the bone; it often form a predominant feature in tubercular inflammation of the bones, etc. I is chiefly by a rarefying osteitis that bone tissue is eroded and destroye in the vicinity of tumors, aneurisms, etc., which exert pressure on th bones. By the same process, the sharp ends of fractured bones may b rounded off as healing proceeds.

When this form of inflammation occurs in cancellous bone tissue the marrow is red or gelatinous, and the bony septa may disappear altogether, so that, in extreme cases, we may have, instead of cancellou bone, a mass of granulation tissue. When the disease occurs in th articular extremity of a bone, the granulating medulla may send littl offshoots through the articular cartilage. These may become fused together, and inflammation of the joint follow. The walls of the shafts of the long bones may be converted into spongy tissue. If, as is sometimes the case, an ossifying periostitis occurs at the same time, the bone is thickened but spongy; or sometimes there are concentric layers of compact bone tissue, separated by rarefied bone.

Condensing Osteitis (Osteo-Sclerosis).—This lesion is characterized by the new formation of bone in the walls of the marrow cavities or Haversian canals. The bone is formed under the influence of the blood-vessels and osteoblasts, as in normal bone formation, but with less regularity. It may result in the conversion of cancellous tissue into compact bone, in the filling-up of the medullary cavity of long bones with more or less dense bone tissue. The compact bone, owing to the filling of its Haversian canals, may become very dense and ivory-like. When the medullary cavities of long bones are involved, the yellow marrow is converted into red marrow by the absorption of fat and increased vascularity. It is frequently associated with ossifying periostitis.

It very frequently follows rarefying osteitis, and under the microscope we can then often see the Howship's lacunæ resulting from the original absorption process filled and covered in with new bone lamellæ (Fig. 201). It is apt to occur in connection with necrosis or some chronic inflammation of adjacent soft parts, but it is sometimes idiopathic or occurs under unknown conditions.

Suppurative Osteitis (Abscess of Bone).—This process occurs usually in the ends of the long bones. It begins with a rarefying osteitis. The medulla undergoes actual suppuration, the bone tissue is destroyed, and a circumscribed cavity is formed in the bone, filled with pus and lined with granulation tissue.

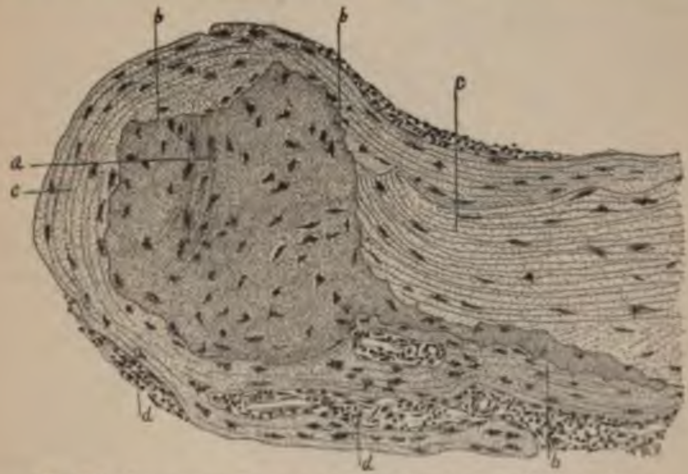

FIG. 201.—CONDENSING OSTEITIS OR OSTEO-SCLEROSIS OF ULNA OF CHILD.

a, fragment of old bone with roughened, sinuous edges; *b*, old Howship's lacunæ covered with more recently formed bone lamellæ.

Less frequently, abscesses are formed in the shaft of a long bone by a circumscribed suppuration of the medulla. These abscesses usually occur in old people. They last for many years, have little tendency to perforation, may gradually enlarge and be accompanied by an ossifying periostitis, so that the bone is expanded. Very rarely acute suppurative osteitis, with rapid formation of an abscess, and perforation, has been observed.

In some cases, instead of abscess, there may be a diffuse infiltration with pus of the Haversian canals or the spaces formed by rarefying osteitis.

Tubercular Osteitis is essentially a rarefying osteitis associated wit the formation of tubercle tissue and cheesy degeneration. The tube cles are sometimes small, scattered, and miliary in form (see Fig. 202 sometimes they unite to form larger and smaller masses. There ma be extensive involvement of the medulla. There may be much simpl granulation tissue or the formation of abscess associated with the pr cess. Condensing osteitis and necrosis are not infrequently presen Tubercular osteitis is often associated with tubercular inflammation o the joints. It is most apt to occur in cancellous bone tissue, and i most common in the bodies of the vertebræ and in the carpal and tars bones. Tubercle bacilli may be found in the tubercular masses, sometimes in considerable numbers.

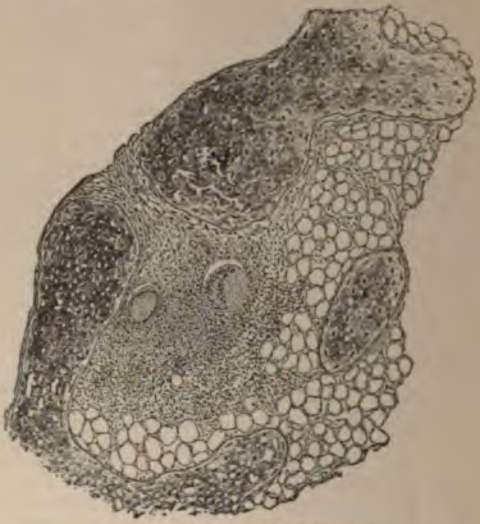

FIG. 202.—TUBERCULAR OSTEITIS.

A miliary tubercle formed in the cancellous tissue near the joint in tubercular arthritis.

Syphilitic Osteitis.—The syphilitic poison may induce one of the above-mentioned varieties of osteitis, or it may produce gummy tumors. The gummatous osteitis usually commences in the periosteum, which becomes thickened and infiltrated with cells, so that there may be a circumscribed thickening of the periosteum, with or without distinct gummata. The vessels which extend from the periosteum into the bone become surrounded by new cellular tissue, which causes an enlargement of the canals. At this stage, if we strip off the periosteum, we drag with it the vessels surrounded by the new cell growth, leaving the bones beneath with numerous small perforations extending inward. As the disease progresses, the gummatous tissue around the vessels continues to increase, and the channels in the bone enlarge by a rarefying osteitis, and coalesce, forming large, irregular defects filled with gummatous tissue. In these masses of new tissue cheesy degeneration and the formation of fibrous tissue occur, giving them the characteristic appearance. In the vicinity of these gumma-filled spaces a condensing osteitis may occur, both in the substance of the bone and on the surface in the form of osteophytes, so that the opening in the bone may be surrounded by an elevated, irregular ring of bone tissue. All this may occur beneath the uninvolved skin, or the skin may participate by a suppurative inflammation, resulting in ulceration. These processes may be circumscribed or involve a large part of a bone. It is not infrequently associated with necrosis of larger and smaller portions of bone. The gummatous tissue may be absorbed and its place be more or less filled with fibrous tissue. Syphilitic osteitis is most frequent in the cranial bones, but may occur elsewhere, as in the sternum, clavicle, tibia and fibula, the ribs, etc.

Congenital Syphilis.—The bones of young children in this condition may occasionally show increased density or evidences of periostitis, or irregular thickenings, particularly of the skull. The researches of Wegner,[1] which have been frequently confirmed by other observers, have shown that exceedingly characteristic changes very uniformly occur in the long bones in still-born or young children who are the victims of hereditary syphilis. These changes are found for the most part along the border zone between the epiphysis and diaphysis. It will be remembered that, in normal ossification of the long bones, the border line between the calcification and ossification zones is narrow, sharply defined, and straight, or gently and evenly curved. In the syphilitic bones, on the contrary, this line is broader, uneven, and presents various modifications, depending upon the stage of the disease. Wegner distinguishes three prominent stages, which, however, merge into one another, so that all intermediate forms may be seen. In the first stage there may be seen, between the cartilage and the new-formed spongy bone, a white or reddish-white zone about two mm. in breadth, with very irregular borders consisting of calcified cartilage, in which the linear groups of cartilage cells are more abundant than normal. In the second stage, the calcified zone, still containing an unusual number of cartilage cells, is broader and still more irregular and less sharply outlined against the ossification zone. The cartilage just beyond it is softer and almost gelatinous, and may contain numerous blood-vessels, islets of connective tissue or of calcification, or irregular ossification. In the third stage, the bone may be pouched out at the sides around the ossification and calcification zones, and the perichondrium and periosteum thickened. The whitish, irregular calcified zone is hard and friable. Between this and the new-formed bone there is an irregular, soft, gray or grayish-yellow zone from two to four mm. in thickness, which forms a loose, readily separated connection between the cartilage and the diaphysis. The white friable zone consists mainly of irregular rows of degenerated and distorted cartilage cells lying in a calcified basement substance, of irregular masses of atypical bone tissue, and of blood-vessels surrounded by variously shaped cells. The soft zone consists of more or less vascular tissue with homogeneous basement substance, and round and spindle-shaped cells. This soft zone is not sharply outlined against the adjoining new-formed spongy bone, which, instead of consisting of the normal marrow spaces with bony lamellæ between them, is largely composed of granulation tissue.

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[1] Virchow's Archiv, Bd. 50, 1870, p. 305.

Different stages of this faulty development may be seen in different bones in the same individual. According to Wegner, the lesion is usually most advanced in the lower end of the femur, then in the lower ends of the leg bones and of the forearm, then in the upper ends of the tibia, femur, and fibula.

Not infrequently there is fatty degeneration of the marrow cells and blood-vessels, giving the marrow a reddish-yellow color. These alterations of the bones may occur, not only in children who have gummata in other parts of the body, but also in those in which other evidences of syphilitic poisoning are absent. So uniform is their occurrence that their presence alone suffices for the establishment of a diagnosis.

OSTEOMYELITIS.

In most of the inflammatory processes which affect the bones, the medulla has an important share, so that many conditions described as osteitis are really osteomyelitis. It is customary, however, to reserve the latter name for those cases in which the medulla is primarily or chiefly involved. Using the word in this sense, we may distinguish a so-called *idiopathic* and a *traumatic osteomyelitis.*

Idiopathic Osteomyelitis.—At the commencement of this disease, which usually begins in the shaft of one of the long bones, there is hyperæmia and œdema of the medulla, so that if the bone be opened the marrow is soft and of a dark-red color. A diffuse suppuration now rapidly ensues, and the marrow becomes streaked or mottled with gray. Occasionally, though not often, larger and smaller abscesses may form in the marrow. The inflammatory areas may be circumscribed and scattered ; or, in the more malignant cases, the entire marrow may become rapidly involved. The cancellous tissue of one or both of the epiphyses usually becomes involved. The disease, however, is not commonly confined to the medullary spaces. The periosteum becomes œdematous and infiltrated with pus, and the surrounding soft parts may become the seat of intense inflammatory changes. Abscesses of the periosteum or surrounding tissues are apt to form. As a result of these changes, necrosis of greater or less portions of the bone may ensue. The medullary cavity may become enlarged as pus accumulates, and the wall of the bone may be broken through, permitting the discharge of pus outward. Sometimes several bones may be involved at once. Secondary involvement of the joints is very frequent. There may be only a serous or purulent exudation; or the acute and destructive inflammatory process may extend to the joint and produce extensive alterations. In young persons, the epiphyses very frequently become separated from the shaft by the destruction of the cartilage which binds them together.

In the severer cases, which are often denominated, *par excellence*, malignant osteomyelitis, the changes may be very rapid and destructive. The medulla becomes broken down and gangrenous; the joints are soon involved; large portions of the bone, sometimes the whole shaft, necrose; the periosteum and surrounding parts become gangrenous; the veins contain thrombi, and pyæmic infarctions and abscesses may form in various parts of the body.

These lesions are, in the large majority of cases, at least due to the presence and action of the pyogenic cocci, the Staphylococcus pyogenes and the Streptococcus pyogenes, and in many of its forms may be regarded as one of the phases of pyæmia.

Traumatic Osteomyelitis.—This form of inflammation may be the result of fracture, amputation, etc. It consists essentially in a more or less diffuse suppurative inflammation of the marrow, variously associated, depending upon the intensity and cause of the disease, with necrosis, gangrene, periostitis, etc. In its more intense and destructive forms, its lesions are similar to those of spontaneous osteomyelitis. Similar bacteria are found in the inflammatory foci, and metastatic pyæmic abscesses may be formed in other parts of the body.

The distinction, then, between the so-called spontaneous or idiopathic infectious osteomyelitis and traumatic infectious osteomyelitis is a superficial one, dependent largely upon the general manner in which the pathogenic bacteria gain access to the body.[1]

In the more chronic forms of osteomyelitis, there is apt to be more or less ossifying periostitis and osteo-sclerosis, and fistulæ may form in the bone, through which the exudations are discharged.

NECROSIS.

By necrosis we understand the death of a larger or smaller portion of bone. This condition is induced by causes which deprive the bone of tis proper vascular supply from the periosteum and medulla. Suppurative periostitis, osteomyelitis, and ostitis, traumatic separation of the periosteum, ulcers of neighboring soft parts, emboli, the action of phosphorus vapor, and diseases, like typhus, which diminish the vitality may cause necrosis. Necrosis is a pure form of gangrene, differing from gangrene of soft parts in that the dead bone has at first, and may retain for a long time, the general outward characters of normal bone; whil in dead soft parts the phenomena of decomposition, under the influenc of bacteria, rapidly ensue, inducing marked complicating appearance in the dead tissue.

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[1] For bibliography, consult *Park*, "Acute Infectious Processes in Bone," Am. Jour. Med. Soc., July, [illegible]

When a portion of bone has died, an inflammation is set up at th dividing line between the dead and living bone. This inflammation ha the characters of a rarefying osteitis (see above), and finally separates th dead from the living bone. The dead bone, or ***sequestrum***, may remain smooth and unaltered, or it may be eroded by the influence of surrounding pus or granulation tissue or osteoclasts. In this way, it is possible for the sequestrum, if it be small, to be entirely absorbed. More frequently there is a production of new bone around the sequestrum, either beneath the periosteum or in the substance of the bone, and this becomes lined with granulation tissue, from which pus may continue to be formed, bathing the sequestrum.

Necrosis may involve the superficial layers, or the entire thickness of the wall of a long bone, or only the spongy tissue and inner layers of the wall, or an entire bone, or a number of different portions of the same bone, but it is most apt to occur in compact bone.

The death and separation of the bone are very soon followed by the growth of new bone to repair the loss. The periosteum, the medulla, and the surrounding soft tissues may all take part in this new growth. The new bone is usually irregular, rough, perforated with openings, through which pus formed around the sequestrum may be discharged. If the sequestrum be removed, healing may occur by the formation of new bone; but the bone is usually more or less distorted by the irregular new ossification.

Phosphorus Necrosis.—Under the influence of phosphorus vapor, periostitis and osteitis, particularly of the jaw, are apt to occur, which usually lead to more or less extensive necrosis, usually associated with prolonged and often extensive suppuration.

CARIES.

Caries of bone is essentially an ulcerative osteitis, resulting in progressive molecular destruction of the bone tissue. It differs from necrosis in that, in the latter, larger and smaller masses of bone die, while in caries the destruction is molecular and gradual. It may occur in connection with any form of osteitis, with periostitis and osteomyelitis, or it may be secondary to inflammatory or destructive processes in the joints or adjacent soft parts. The depressed surfaces of bones in which caries is progressing are rough and more or less finely jagged, and may be covered with granulations. The minute changes by which ulceration and destruction of the bone are produced in caries are somewhat analogous with those in rarefying osteitis, but there are marked degenerative changes in the bone cells, which may become fatty or converted into a granular material. Moreover, the basement substance of the bone, instead of being absorbed, may disintegrate, with the formation of larger and smaller masses of detritus. Sometimes the lime salts are removed from the basement substance, which is converted into atypical fibrillar tissue and fatty and granular detritus. Very extensive suppurations and necrosis may be associated with caries.

Long-continued caries, especially in badly nourished individuals, is apt to become complicated with tubercular inflammation.

There is very little tendency to spontaneous healing in caries, but it may occur, and the defects produced may be more or less supplied by means of new-formed bone.

RACHITIS (RICKETS).

Rickets is a disease affecting the development of bone, preventing its proper ossification. The disease usually occurs during the first two years of life, but may be congenital, or may occur as late as the twelfth year.

The physiological growth of bones depends upon three conditions. They grow in length by the production of bone in the cartilage between the epiphysis and diaphysis; in thickness, by the growth of bone from the inner layers of the periosteum. At the same time the medullary canal is enlarged, in proportion to the growth of the bone, by the disappearance of the inner layers of bone.

In rickets, these three conditions are abnormally affected. The cartilaginous and subperiosteal cell growth, which precedes ossification, goes on with increased rapidity and exuberance, and in an irregular manner, both between the epiphyses and diaphyses, and beneath the periosteum, while the actual ossification is imperfect, irregular, or wanting. At the same time the dilatation of the medullary cavity goes on irregularly and often to an excessive degree.

If we examine microscopically the region between the epiphysis and diaphysis (Fig. 203), we find that the cartilage cells are not regularly arranged in rows along a definite zone in advance of the line of ossification, as in normal development, but that there is an irregular heaping-up of cartilage cells, sometimes in rows, sometimes not, over an ill defined and irregular area. The zone of calcification also, instead of being narrow, regular, and sharply defined, is quite lacking in uniformity. Areas of calcification may be isolated in the region of proliferating cartilage cells, or calcification may be altogether absent over considerable areas.

Corresponding to these irregularities, the ossification zone is als irregular. New formed bone and marrow cavities containing bloo vessels may lie in the midst of the cartilage, or masses of cartilage ma lie deep in the region which should be completely ossified. In othe places, it seems as if the cartilage tissue were directly converted into a ill-formed bone tissue by metaplasia or direct transformation. It wi readily be seen from this that the medullary spaces of the new-forme bone are irregular, and this abnormality is enhanced by the prematur intramedullary absorption of the bone.

The same sort of irregularity in the bone formation may be seen beneath the periosteum. An excessive proliferation of cells in the inne layers of the periosteum, the irregular calcification which occurs abou them, and the absence of uniformity in the elaboration of ill-structured bone, conspire to produce an irregular, spongy bone tissue instead of the compact, lamellated tissue which is so necessary here for the solidity of the structure. The increased cell growth between the epiphyses and diaphyses produces the peculiar knobby swellings which are characteristic of rickets. At the same time the medullary cavity increases rapidly in size, and the inner layers of the bone become spongy. The medulla may be congested, and fat, if it has formed, may be absorbed, and a modified form of osteitis may ensue.

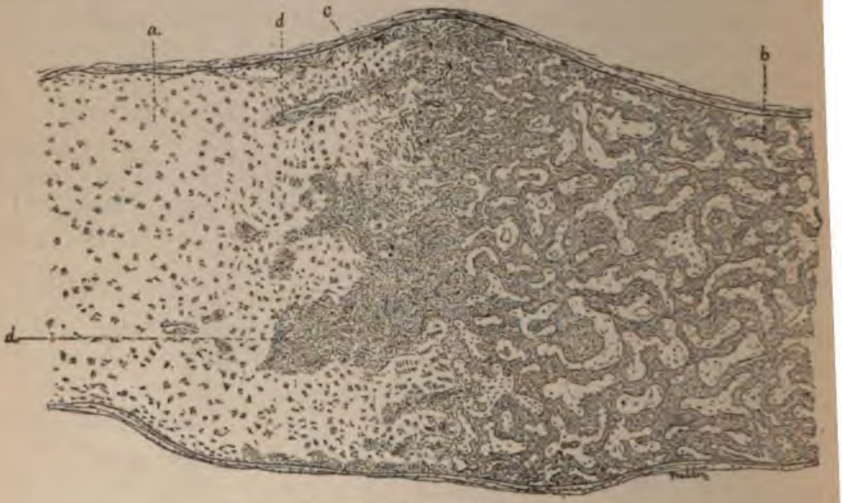

FIG. 203.—RACHITIC BONE.
Showing ossification zone in a longitudinal section of a rib.

The result of these processes is that the bones do not possess solidity and cannot resist the traction of the muscles or outside pressure. The epiphyses may be displaced or bent, especially in the ribs, less frequently in the long bones. The long bones and the pelvic bones may be bent into a variety of forms. Incomplete fractures are not infrequent. Complete fractures do not usually occur until the later stages of the disease, when the bones have become more solid. In the head, the cranium may be unnaturally large for the size of the face; the fontanelles and sutures may remain open; the bones may be soft, porous, and hyperæmic, while at their edges there may be rough, bony projections beneath the pericranium. Sometimes, especially in the occipital bone, there are rounded defects in the bone, filled only with a fibrous membrane; this constitutes one of the forms of so-called *craniotabes*.

It does not fall within the scope of this work to describe the various deformities which may occur as a result of this disease. The familiar pigeon breast; the rows of knobs along the sides of the chest from bending and dilatation of the ribs at the point of junction of cartilage and bone; the knock-knee, bow-legs, spinal curvatures, etc., may all be the result of rachitic weakening of the bones.

After a time, the rachitic process may stop and the bones take on a more normal character. The porous bone tissue becomes compact and even unnaturally dense; the swellings at the epiphyses disappear; many of the deformed bones may become of a normal shape. In severe cases, however, the deformities continue through life; especially is there a cessation of the growth of the bones in their long axis, so that the persons affected are dwarfed.

The disease may have an acute or a chronic character. The acute form begins usually during the first six months of life. The children are apt to suffer from vomiting, diarrhœa, profuse sweating, chronic bronchitis and pneumonia, general anæmia, and wasting. They either die or the rachitic process is gradually developed. The chronic form is seen in older children, and often in those apparently healthy. The changes in the bones may take place without any constitutional symptoms, though there is often catarrhal bronchitis, pneumonia, and anæmia.

OSTEOMALACIA.

This lesion consists in the softening of fully formed hard bone tissue by the removal of its inorganic salts. It is to be clearly distinguished from rickets, whose lesions are due to a faulty development of bone, although in certain external characters the two diseases sometimes present considerable similarity. Osteomalacia usually occurs in adults, most frequently in females during pregnancy and after parturition; more rarely it occurs in males, and in females unassociated with the above conditions. Its cause is not known.

Microscopical examination shows that the decalcification occurs first in the periphery of the Haversian canals and in the inner layers of the walls of the marrow spaces. As the salts of lime are removed, the basement substance at first remains as a finely fibrillated material, still preserving the original lamellation. The bone cells may be changed in shape or degenerated. After a time, the decalcified tissue may disintegrate and be absorbed, and its place occupied by new-formed marrow or granulation tissue. As the disease goes on, the marrow tissue is congested and red, the fat absorbed, and there is a great accumulation of small spheroidal cells; or the marrow may assume a gelatinous appearance. The decalcification and absorption of the bone from within may proceed so far that the bony substance in the cancellous tissue almost entirely disappears, and the compact bone is reduced to a thin, soft, decalcified tissue. The disease is not always continuously progressive, but may be subject to temporary cessation.

As a result of this softened condition of the bones, the weight of the body and the actions of the muscles may induce a series of deformities which are sometimes excessive: curvatures of the spine, complete and incomplete fractures of the bones, distortions of the pelvis, sternum, etc.

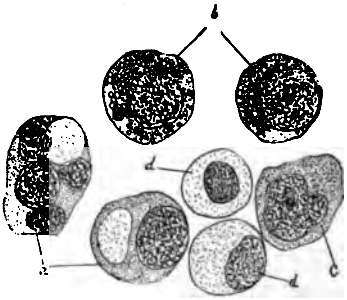

FIG. 204.—CELLS FROM THE MARROW OF FEMUR IN LEUKÆMIA.

a, cells containing red blood-cells or fragments of red blood-cells; *b*, small spheroidal cells in a condition of fatty degeneration; *c*, cells like the last, but not fatty; *d*, nucleated red blood-cells.

There is a tendency in this disease to a general involvement of the bones, but the changes are sometimes confined to single bones or groups of bones. The cranium is rarely much affected.

ALTERATIONS OF THE BONE MARROW IN LEUKÆMIA AND ANÆMIA.

In certain forms of leukæmia, the marrow of the bones is very markedly altered. The change consists mainly in an accumulation in the marrow tissue of small spheroidal cells, often in a condition of fatty degeneration, which lie in the meshes of reticular connective tissue and in and along the walls of the blood-vessels. There may also be absorption of the fat, and sometimes enlargement of the marrow cavity from absorption of the bone. The marrow cavity may also contain, intermingled with its other elements, nucleated red blood-cells, small spheroidal cells which contain red blood-cells (Fig. 204), and not infrequently considerable numbers of small octahedral crystals (called Charcot's crystals).

The degree to which this accumulation of cells occurs varies much in different cases, and the gross appearances of the marrow are consequently very variable. In some cases, the marrow is soft and has a uniform red appearance, or it is variously mottled with gray and red. Occasionally circumscribed hæmorrhages are seen. In another class of cases, in which the cell accumulation is more excessive, the marrow may be gray, grayish yellow, or puriform in appearance.

These changes may occur in the central marrow cavity, as well as in the marrow spaces of the spongy bone. They may be present in several or many of the bones. They are usually accompanied by analogous changes in the spleen and lymph nodes.

In certain cases of acute and chronic *anæmia*, particularly in the pernicious and progressive varieties, the marrow, especially of the larger long bones, may lose its yellow color from absorption of the fat, and become red. Microscopical examination of the marrow under these conditions shows considerable increase of small spheroidal cells, and sometimes an abundance of developing nucleated red blood-cells and Charcot's crystals.

In many of the acute infectious diseases, typhus and typhoid fever, ulcerative endocarditis, recurrent fever, etc , the bone marrow has been found hyperæmic and containing an unusual number of small spheroidal cells.

All of these lesions of the marrow, although our knowledge of them is still very incomplete, together with what is known of the physiological functions of the marrow, point to a close relationship between the marrow and the spleen and lymph nodes as blood-producing organs.[1]

ATROPHY.

In old age or in senile conditions, the bones may become atrophied by the absorption of the hard tissue; the medullary spaces are enlarged, the marrow tissue contains less fat and is often gelatinous in appearance. As the result of the lack of use, or from any cause which interferes with the nutrition of the bone, such as paralysis of the muscles or diseases of the joints, the bones may atrophy. In connection with atrophy there may be an ossifying periostitis, which results in making the bone look even larger than normal. Many of the conditions commonly called atrophy, such as the erosions of bones from tumors, etc., pressing upon them, are really due to a rarefying osteitis.

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[1] The literature of the researches on the diseases of the spleen, which are important in this connection, may be found in part in *Orth's* "Lehrbuch der speciellen pathologischen Anatomie." Berlin, 1883. Erste Lieferung, p. 119 et seq.

The bones, sometimes as the result of atrophy and sometimes fro causes which we do not understand, are unusually brittle and liable t fracture. This disposition is sometimes hereditary.

TUMORS.

Tumors of the bone may involve either the periosteum, the compac bone, or the medulla, or, as is more frequently the case, two or more o these structures are involved at once. Tumors of the bone are usuall accompanied by various secondary and sometimes very marked altera tions of the bone tissue, osteo-porosis, osteo-sclerosis, ossifying periostitis etc. The new growths are very apt to undergo calcification and ossifi cation.

Fibromata may grow either from the periosteum or medulla. Thei most common seat is in the periosteum of the bones of the head and face. They are apt to form polypoid tumors projecting into the posterior nares, pharynx, mouth, and antrum of Highmore. Central fibromata, *i.e.*, those growing from the medulla, are rare. They usually occur in the lower jaw, but have been found in the ends of the long bones, the phalanges of the fingers, and the vertebræ. The fibromata may calcify or ossify, contain cysts, and not infrequently occur in combination with sarcoma.

Myxomata are of occasional occurrence in bone.

Osteomata.—New formations of bone, as a result of inflammatory processes, are, as we have already seen, of frequent occurrence in bone, and although not, strictly speaking, tumors, some of their forms are very closely allied to them, and they may therefore be conveniently mentioned here. New growths of bone which arise from the surfaces are called *exostoses* or *enostoses*, according to their origin from the external surface or interior of the bone. They may contain all the constituents of normal bone: bone, medulla, vessels, periosteum, and cartilage. The new bone may be compact and like ivory, or spongy, or contain large cavities filled with marrow.

The shape of exostoses varies greatly; they may be in the form of sharp, narrow spiculæ and processes, and, occurring in connection with periostitis, are called *osteophytes*. They may be polypoid in shape or form rounded tumors with a broad base. They may form a general enlargement of the bone, with much roughening of the surface; this condition is often called *hyperostosis*.

The bone beneath these new growths may be normal, or sclerosed, or rarefied, or the medullary cavity of the bone may communicate with that of the exostosis. Exostoses are usually developed from the periosteum, sometimes in the insertion of tendons and ligaments. They are very frequently multiple, and may occur at all ages, even during uterine life.

Enostoses are developed in the interior of bones from the medulla. They may increase in size, with absorption of the surrounding bone, until they project from the surface like exostoses. Their most frequent situation is in the bones of the cranium and face.

Chondromata.—These tumors may be single or multiple, and most frequently grow from the interior of the bone, but sometimes from the periosteum. They are prone to form various combinations with other forms of tumors, as fibroma, myxoma, sarcoma, etc. They are frequently congenital and are most common in young people. They occur most frequently in the bones of the hand and foot.

There is a form of chondroma, called *osteoid chondroma*, which develops beneath the periosteum, most frequently in the femur and tibia near the knee joint, forming a club-shaped enlargement of the bone. The characteristics of the tissue composing these tumors are, that it resembles somewhat the immature bone tissue which is seen beneath the periosteum in developing bone. It differs from cartilage in the irregular shape of its cells, in the fibrillation and density of the basement substance, and in its general vascularity. On the other hand, it has not the inorganic contents or appearance of true bone. It resembles considerably the callous tissue forming about fractures of the bones. It may, however, and most frequently does, become converted, in some parts of the tumor, into true bone. On the other hand, combinations with sarcomatous tissue are of frequent occurrence (see below).

Sarcoma.—This form of tumor is especially common in the bones. It grows from the inner layers of the periosteum or from the medulla, so that we may distinguish a *periosteal* and a *myelogenic* sarcoma. Sometimes the tumor attacks the bone itself so early that it is impossible to say whether the tumor began in the periosteum or in the medulla. There is also a variety which grows close to the outside of the periosteum and becomes connected with it—*parosteal* sarcoma.

The *periosteal sarcomata* usually belong to the varieties fibro-, myxo-, chondro-, and osteo-sarcoma, more rarely to the medullary variety. They commence from the inner layers of the periosteum, pushing this membrane outward. After a time the periosteum is attacked, and the tumor invades the surrounding soft parts. The bone beneath may remain normal, or may be eroded and gradually disappear until the tumor is continuous with the medulla. Portions of the tumor may be calcified, or a growth of new bone may accompany its growth. The new bone usually takes the form of plates, or spiculæ, radiating outward. The minute anatomy of these tumors is very variable. The simplest—the fibro-sarcomata—are composed of fusiform, round, stellate, and sometimes giant cells (myeloplaxes), in varying proportions, packed closely in a fibrous stroma. In the medullary form, the stroma is diminished to a minimum and the round cells are most numerous. In the chondro- and myxo-sarcoma, the basement substance may be hyalin or mucous, and the cells follow the type of cartilage and mucous tissu more or less closely. There is a mixed form of tumor, called *osteoi sarcoma*, which is very apt to spread and to form metastases. Th growth consists in part of tissue corresponding to fibro-sarcoma an round-celled sarcoma. In addition to this, there occurs, in greater o less quantity, immature bone tissue, called osteoid tissue, which may i part become calcified, the calcification usually occurring in the centra portions, leaving a softer peripheral zone. This form of tumor is mos apt to occur at the ends of the long bones, and may form tumors o large size. It is often called, on account of its tendency to spread and to form metastases, *malignant osteoma* or *osteoid cancer.*

Myelogenic sarcomata commence in the medulla and may grow rapidly. The bone surrounding them is destroyed, and they project as rounded tumors. Most frequently new bone is formed beneath the periosteum, so that the tumor is inclosed in a thin, bony shell; sometimes there are also plates of bone in the tumor; sometimes the periosteum is unaltered; sometimes it is perforated, and the tumor invades the surrounding soft parts. The tumors are frequently very soft, vascular, and hæmorrhagic in parts, or may inclose cysts filled with tumor detritus and blood. They are usually of the spindle or round-celled variety, and not infrequently contain giant cells.

The *parosteal sarcomata* resemble the periosteal, but they appear to grow from the outer layers of the periosteum. They may be as firmly connected with the bone as the periosteal form. The periosteum may remain intact between the tumor and the bone, or it may disappear and leave them in apposition.

Angiomata and Aneurism of Bone.—A very large number of the tumors which have been described under these names are really sarcomata, or other tumors which happened to be very vascular. Some authors, indeed, are disposed to deny altogether the existence of real vascular tumors in bones. There are, however, reliable cases of cavernous angiomata growing between the periosteum and bone, and intimately connected with the latter. Whether myelogenic angiomata occur is doubtful. There are several cases described of cavities filled with blood in the interior of bones, which it is difficult to interpret. They have mostly been found in the head of the tibia. They are said to have consisted of single sacs composed of thickened periosteum, lined with plates of bone, and filled with fluid and clotted blood. No large vessels communicated with the sacs, but their walls were covered with a rich vascular plexus, branches of which opened into the cavity of the sac.

Carcinomata.—Primary carcinomata are of very doubtful occurrence in the bones. Most of the structures thus named have doubtless been sarcomata. Secondary carcinomata, on the other hand, as a result of metastases or local extension, are of not infrequent occurrence and present various structural forms. Metastatic carcinomata may occur in the bones of various parts of the body at the same time, and are most apt to be secondary to carcinoma of the mamma.

Cysts.—These most frequently occur in the maxillary bones, doubtless in connection with the teeth. They may be unilocular or multilocular, and contain clear serum or a mucous or brown fluid, and sometimes cholestearin. They may be lined with epithelium. They begin in the interior of the bone, and, as they increase in size, expand it until they may be covered with only a thin shell of bone. They may reach a large size, even as large as a child's head.

Dermoid Cysts are occasionally found in connection with the bones, particularly of the skull.

PARASITES.

Echinococcus and *cysticercus* are of rather rare occurrence in the bones; the former is most frequently found.

DISEASES OF THE JOINTS.

For a description of the dislocations, misplacements, and injuries of the joints, we refer to works on surgery.

INFLAMMATION.

Acute Arthritis.—The earlier stages of acute inflammation of the synovial membranes are better known from experiments on animals than from post-mortem examinations. The first changes are swelling and congestion of the membrane, with increased growth and desquamation of epithelium, and infiltration of the membrane with lymphoid cells. These conditions are soon followed by an exudation. The exudation may be a clear serum, in which epithelial cells, lymphoid cells, and sometimes blood will be found. Or flocculi of fibrin may float in the serum, or the fibrin may be in excess and the serum nearly absent. Or there is an excessive production of lymphoid cells, and the synovial sac is filled with pus.

In *Serous Arthritis,* the accumulation of serum within the synovial sac is the most prominent lesion. The disease may terminate in recovery, or become chronic, or pass into the suppurative form. It may be caused by contusions, penetrating wounds, gonorrhœa, rheumatism, or it may occur without evident cause.

Sero-fibrinous Arthritis may occur under the same conditions as those which lead to simple serous inflammation. The fibrin may be present largely as flocculi in the serum, or it may form false membranes over the surfaces of the joint.

Purulent Arthritis may follow or be associated with the above forms of inflammation. The synovial membrane is thickened and cloudy, and there may be but a moderate amount of pus in the joint, and a slight degree of infiltration of the synovial membrane with pus cells. Under these conditions resolution may readily occur.

In other cases, the accumulation of pus in the cavity may be great, the synovial membrane and its surrounding tissue densely infiltrated with pus cells. Under these conditions, granulation tissue is apt to be found and the cartilages of the joints are apt to become involved. There are swelling and proliferation or degeneration of the cartilage cells; the basement substance becomes disintegrated, ulcerates, and exposes the bone, in which osteitis, caries, rarefaction, etc., may occur. The new-formed granulation tissue may penetrate the cartilage, absorbing the basement substance, and by metaplasia the cartilage tissue may be converted into embryonal or granulation tissue. The pus may break through the capsule of the joint and form large abscesses in the adjacent soft parts. Sometimes the inflammation is not only suppurative but gangrenous, and runs a rapidly fatal course. The synovial membrane, articular cartilages, and ends of the bone, all undergo a rapid suppuration and gangrene. Pyæmia and septicæmia, small-pox, measles, scarlet fever, diphtheria, mumps, typhus fever, glanders, the puerperal condition, exposure to cold, penetrating wounds, and injuries, may all give rise to purulent synovitis.

Chronic Arthritis may begin as such or it may be the result of previous acute inflammation. There is an increase of fluid in the joint. This fluid is thin and serous, or is thickened with flocculi of fibrin and epithelial and lymphoid cells, or is thick, syrupy, or even gelatinous. The synovial membrane is at first congested, its tufts prominent. Later it becomes thickened, sclerosed, and anæmic; the epithelium is destroyed, and the tufts become large and projecting. From the distention of the capsule there may be subluxations or luxations of the joint, or the capsule may be ruptured.

Chronic Rheumatic Arthritis is most common in elderly persons, usually affecting several joints and advancing slowly and steadily. There is a chronic thickening of the synovial membrane and the fibrous tissue adjacent to it. Fluid accumulations are not common. The articular cartilages are apt to degenerate or ossify, or become softened and fibrillated, and they may disappear. The contracting synovial membranes and fibrous tissue render the joints stiff, and may cause considerable deformity. Not infrequently fibrous and bony anchyloses are formed between the ends of the bones.

Arthritis deformans.—This name has been applied to a variety of chronic inflammation of the joints which, combined with degeneration of parts of the joint and the new formation of bone, may result in marked deformities of the part.

It usually occurs in elderly persons, and is apt to involve several joints, most frequently the hip, knee, fingers, and feet. It may be idiopathic, or due to rheumatism or to injuries, or follow an acute arthritis. The capsules of the affected joints are thickened and sclerosed. The synovial fluid is at first increased in quantity; later, diminished and thickened. The tufts of the synovial membrane become much enlarged and vascular; they may be converted into cartilage. Sometimes the capsule becomes ossified. The new bone grows from the edge of the cartilage within the capsule, and its articular surface is covered with cartilage. The articular cartilages are much changed. The basement substance splits into tufts, while the cartilage cells are increased in number. Or the basement substance becomes fibrous; or it is split into lamellæ and the cartilage cells are multiplied; or there is fatty degeneration and atrophy.

As a result of these changes, larger or smaller portions of the cartilage are destroyed, and the bone beneath is laid bare. The exposed bone may become compact and of an ivory smoothness. The ends of the bones are much deformed. They are flattened and made broader by irregular new growths of bone, while at the same time they atrophy. The new growth of bone starts from the articular cartilages. The cartilage cells increase in number, and the basement substance in quantity. This growth is most excessive at the edge of the cartilage, so that a projecting rim is formed there. This projecting rim may ossify next the bone, and at the same time new cartilage may form on its surface, so that we may find large masses of bone covered with cartilage. All these changes occur in various combinations and sequences, so that joints in this condition present the greatest variety of appearances.

Arthritis uritica (Gouty Arthritis).—This disease is characterized by the deposit of salts of uric acid in the cartilages, bones, and ligaments, and also in the cavities of joints. The deposits may be in the form of stellate masses of acicular crystals in and about the cartilage cells or in the basement substance; or they may be deposited in the fibrillar connective-tissue structures of the joint in single crystals, or in the subcutaneous tissue about the joint as white concretions. The deposits may occur in repeated attacks of the disease, and are accompanied by acute inflammatory changes. They may lead to various forms of chronic inflammation of the joints.

Tubercular Arthritis (Chronic Fungous Arthritis; Strumous Arthritis).—This disease may commence in the joint itself, or be transmitted to it from a tubercular inflammation of the bone. It is characterized by the formation of granulation tissue containing tubercles, sometimes in great quantity, and usually associated with secondary inflammatory and degenerative changes of surrounding parts. According to the prominence of one or other of these secondary alterations, several forms of tubercular arthritis may be distinguished. If there is an excessive growth of granulation tissue without much suppuration, this constitutes a *fungous form*. Sometimes there is extensive *suppuration*, so that the cavity of the joint may be filled with pus, which may be discharged through openings in the skin; or there may be more or less extensive formation of abscesses, or infiltration of the soft parts about the joint with pus. In other cases, there is a predominant tendency to breaking down of the new-formed tubercular tissue and of the tissues of the joint—*ulcerative form*. The cartilage basement substance may become split into fragments and the cells degenerate, and thus deep and destructive ulcers of the cartilage be formed. Or the granulation tissue may work its way through the cartilage into the bone beneath. by absorption of the basement substance of the cartilage, with or without proliferation of its cells. Caries and necrosis of the underlying bone may lead to extensive destruction. Hand-in-hand with these alterations subperiosteal new formation of bone may occur, or sclerosis of the adjacent bone tissue. There may also be a great increase of fibrous tissue about the joint. Tubercle bacilli may be found in the tubercular tissue and in the exudations.

This disease is most common in children and young persons. The so-called scrofulous diathesis is said to dispose to it, but local injuries are frequently the predisposing factors. It is most common in the large joints. It may occur in connection with tubercular inflammation in other parts of the body, but it is frequently quite local, and may remain so for a very long time or permanently, since general infection from tubercular arthritis is comparatively infrequent.

The disease always runs a very chronic course and may destroy the patient's life. If recovery takes place before the cartilages and bones are involved, the joint is preserved; but it may be stiffened, or even immovable, from the contraction of the new fibrous tissue around it. If the cartilages and bones are diseased, the joint is destroyed, and either bony or fibrous anchylosis results. Sometimes from the change in the articulating surfaces, and the contraction of the muscles and the new fibrous tissue, partial or complete dislocations are produced.

Occasionally miliary tubercles occur in the synovial membranes in cases of general miliary tuberculosis, with but little accompanying simple inflammatory change.

TUMORS.

Secondary tumors of the joints as a result of local extension from the adjacent parts are not uncommon, and the tumors may be of various kinds. Primary tumors of the joints, on the contrary, are not very common.

Lipoma.—A new growth of fatty tissue may begin in the other portions of the synovial membrane, push this inward, and project into the joint in a mass of tufts—lipoma arborescens.

Fibroma occurs as an hypertrophy of the little tufts and fringes of the synovial membrane. In this way, large polypoid and dendritic bodies are formed. The pedicles of these growths may atrophy and even disappear, so that the growths are left free in the cavities of the joints.

Corpora aliena Articulorum (Loose Cartilages in the Joints).—This name is given to bodies, of various structure and origin, which are found free or attached by slender pedicles in the cavities of the joints.

They are most frequently found in the knee; next in order of frequency in the elbow, hips, ankle, shoulder, and maxillary joints. They may be single or in hundreds. Their size varies from that of a pin's head to that of the patella. They are polypoid, rounded, egg-shaped, or almond-shaped; their surface is smooth or faceted, or rough and mulberry-like. They are composed of fibrous tissue, cartilage, and bone in various proportions.

These bodies are formed in different ways.

1. By hypertrophy of the synovial tufts and production of cartilage and bone in them.

2. More frequently by a change into cartilage of portions of the synovial membrane. Small, flat plates of cartilage form on the inner surface of the synovial membrane, and these increase in size and their outer layers ossify. They may remain fixed in the synovial membrane, or they project and become detached from it, and they then appear as flattened concave bodies composed of bone covered with cartilage on one side.

3. The growth of cartilage and bone begins in the outer layers of the synovial membrane or in the periosteum near the joint. The new growth pushes the synovial membrane inward, and projects into the joint as a polypoid body covered with the inner layers of the synovial membrane. Later the membrane atrophies, and the growth becomes free in the joint.

4. There may be cartilaginous outgrowths from the edges of the articular cartilage.

5. Rarely portions of the articular cartilages may be detached by violence or disease; or fibrinous and other concretions may result from arthritis, or under conditions which we do not understand.

MUSCLE.

LESIONS OF VOLUNTARY STRIATED MUSCLE.

Hæmorrhage.—This may occur as a result of mechanical injury; from rupture of the fibres by convulsive contraction, as in tetanus; or it may occur when the muscle fibres are degenerated, as in typhoid fever; or in connection with certain general diseases, as scurvy, purpura, hæmorrhagic diathesis, septicæmia, etc. The blood is usually readily absorbed.

Embolic Infarction of Muscles in connection with heart disease has been described in a few cases, but it is rare.

Wounds and Rupture.—When the muscle fibres are severed by wounds or rupture, there is more or less degeneration of the divided fibres, and the wound may heal by the production of granulation tissue, which gradually becomes converted into cicatricial tissue, thus binding the severed parts together. In some cases there is a new formation of muscle fibres, which penetrate the cicatrix and establish muscular connection between the parts. When the wound does not gape, so that the severed ends are not much separated, there may be, it would seem, a direct re-establishment of muscular continuity by new development of muscle, without the formation of much new connective tissue.

The exact way in which muscle fibres are regenerated is yet somewhat uncertain. In many cases, there seems to be a proliferation of the so-called muscle corpuscles, leading to the formation of elongated cells or strings of cells, which are gradually converted into striated muscle. In some cases, the appearances would seem to indicate that connective-tissue cells, and perhaps white blood cells, may participate in the formation of new muscle fibres, but this is not certain.

INFLAMMATION.

Suppurative Myositis.—In the early stages of the lesion we find the muscle hyperæmic and œdematous and the interstitial tissue more or less infiltrated with small spheroidal cells, doubtless the result of emi-

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[1] For literature of muscle regeneration consult [illegible] Arch. für exp. Pathologie u. Pharm., Bd. 25, Heft 5 und 6, p. 415, 1889.

gration. If the inflammation becomes intense, there may be an excessive accumulation of pus cells, either diffusely in the interstitial tissue or in larger and smaller masses. Hand-in-hand with this cell accumulation occur degenerative changes in the muscle fibres. By pressure their nutrition is interfered with, and they undergo granular, fatty, or hyaline degeneration. They may completely disintegrate and gangrene may occur, so that larger and smaller masses of the infiltrated muscle tissue become soft, foul-smelling, and converted into a mass of detritus in which but little muscle structure can be detected, and which is intermingled with bacteria. In other cases there may be larger and smaller abscesses formed in the muscle, the muscle tissue itself either degenerating and disintegrating and mixing with the contents of the abscess, or being pressed aside and undergoing atrophy and degeneration. In some cases when the formation of pus is moderate in amount, there may be restoration by formation of granulation tissue between the muscle fibres. This becomes gradually dense and firm, and leads to more or less atrophy of the muscle fibres by pressure.

Acute suppurative myositis may accompany wounds; it is very common in acute phlegmonous inflammations of the skin and subcutaneous tissue, and often accompanies acute infectious diseases, such as pyæmia, erysipelas, etc. In many cases, colonies of micrococci are present in the inflammatory foci. It is not infrequently seen in the muscles adjacent to the inflamed mucous membranes in diphtheria.

Acute Parenchymatous Myositis.—A few cases of this disease have been described, in which, without lesion of the nervous system, certain groups of muscles, with the occurrence of fever and pain, become swollen, in some cases beset with small hæmorrhages, soft, mottled with yellowish-white patches. Microscopically the muscle fibres showed granular and fatty, or in some cases waxy, degeneration. The cause of this lesion is not known. In one case, the muscles of the legs were thus affected in a woman who died in the first week after delivery, with fever and pain in the legs, and the lesion was conjecturally of infectious origin.[1]

Chronic Interstitial Myositis.—In this lesion there is a new formation of connective tissue between the muscle fibres or bundles of fibres. This new tissue is sometimes very cellular, resembling granulation tissue, and this probably represents an early stage of the disease. In other cases (Fig. 205), we find dense cicatricial tissue crowding the muscle fibres apart, inducing atrophy in them, and sometimes causing their complete destruction. This lesion, which is the analogue of chronic interstitial inflammation of the internal organs, may occur in muscles which are adjacent to other parts which are the seat of chronic inflammatory processes. It may occur in muscles which are not used. The new formation of connective tissue would in some cases seem to be secondary to atrophy of the muscle fibres.

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[1] Consult *Eisenlohr*, Centralblatt für Nervenheilkunde, 1, 1879 ; ***Marchand***, Breslauer Aerztliche Zeitschrift, 21, 1880.

Myositis ossificans.—Under conditions and for reasons which we do not understand, there occasionally occurs, usually in young persons, a new formation of bone tissue in the interstitial tissue of muscles, in the tendons, ligaments, fasciæ, and aponeuroses. This sometimes apparently starts as outgrowths from the periosteum, sometimes not. The bone formations are apt to commence about the neck and back, and may become very widespread over the body. So far as the muscles are concerned, there is usually an increase of connective tissue between the fibres and bundles, in which new bone is formed, usually in elongated and sometimes in spicula-like masses. The muscle fibres undergo secondarily a greater or less degree of atrophy or degeneration. There may be fatty infiltration between the fibres, and various deformities are produced by the shortening and progressive immobility of the affected parts.[1]

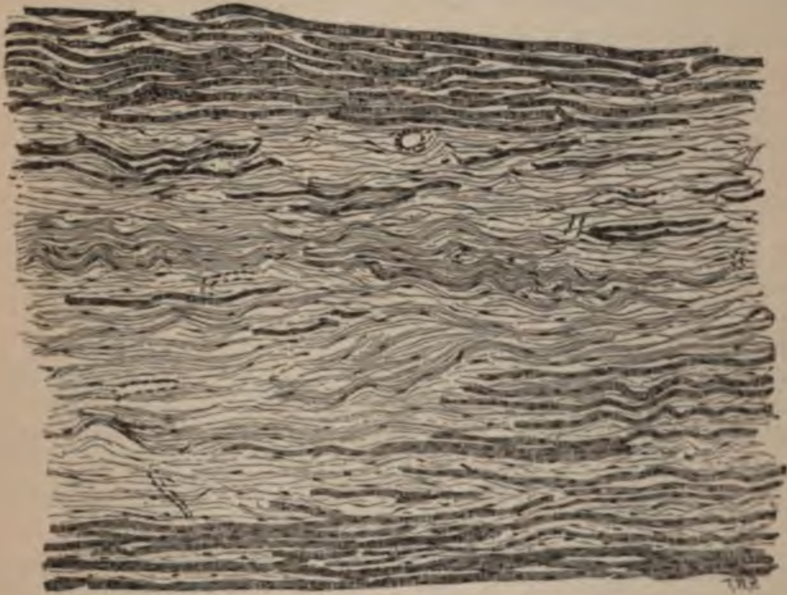

FIG. 235.—CHRONIC INTERSTITIAL MYOSITIS.

The connective tissue is dense in texture, and the muscle fibres are atrophied and partially destroyed.

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[1] The literature of Myositis ossificans may be found, together with a description of some interesting cases, in an article by *Mays* in Virch. Archiv, Bd. 74, p. 145.

While the above disease is a progressive and frequently a general one, there may be new formation of bone in muscle as a result of prolonged or repeated mechanical irritation. Thus in the adductors of the thigh, in persons who are constantly in the saddle, or in the deltoid muscle of soldiers, who strike this part with their weapons in drill, there may be a formation of bone.

Gummata and occasionally *tubercles* occur in the connective tissue of muscle.

DEGENERATIVE CHANGES IN THE MUSCLES.

Simple Atrophy.—This may occur in old age, in prolonged exhausting diseases, or as a result of pressure from a foreign body, tumors, etc. The muscle fibres grow narrower, the degree of narrowing frequently varying considerably in different parts. They usually retain the striations, but these may be obscured by degenerative changes. The sarcolemma may become thickened, and there may be a considerable increase in connective tissue between the muscle fibres and bundles.

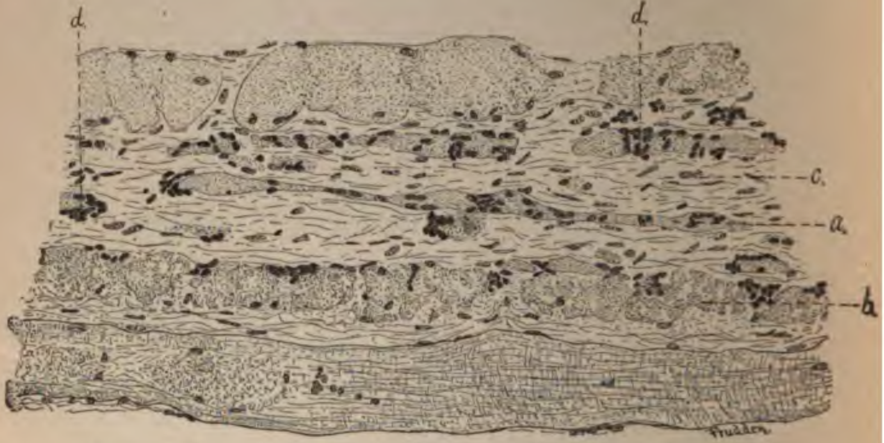

FIG. 206.—PROGRESSIVE MUSCULAR ATROPHY (Soleus muscle, longitudinal section).
a, atrophied muscle fibre; *b*, degenerated muscle fibre; *c*, interstitial tissue; *d*, clusters of proliferated muscle nuclei.

Progressive Muscular Atrophy.—This lesion consists essentially in a combination of simple or degenerative atrophy of the muscle fibres with chronic interstitial inflammation, and is sometimes associated with proliferative changes in the muscle nuclei. In the earlier stages of the disease, the muscles may be pale and soft, but exhibit otherwise to the naked eye but little alteration. Gradually, however, the muscle substance becomes replaced by connective tissue, so that in marked and advanced cases the muscles are converted into fibrous bands or cords, whose cicatricial contraction may induce great deformities.

Microscopical examination shows in the early stages of the disease a proliferation of cells in the interstitial tissue, so that this may have the appearance of granulation or embryonal tissue; also in some cases marked proliferative changes in the muscle nuclei (Fig. 206), leading to the formation of new cells which may more or less replace the contractile substance within the sarcolemma. The new interstitial tissue increases in quantity and grows denser, and may crowd the muscle fibres apart (Fig. 207). The walls of the blood-vessels may also become thickened. Hand-in-hand with these interstitial alterations the atrophy of the muscle fibres proceeds. These may simply grow narrower, retaining their striations; or they may split up into longitudinal fibrillæ; or transversely into discoid masses, and in this condition disappear. In other cases, a certain amount of fatty or hyaline degeneration may be present. These degenerative and proliferative changes do not, as a rule, occur uniformly in the affected muscles, but some parts are affected earlier and more markedly than others. The atrophied muscle may be replaced by fat.

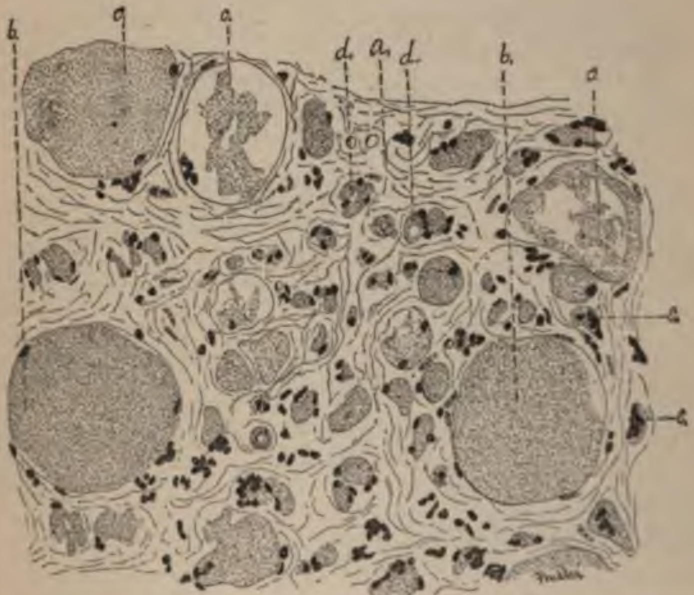

FIG. 207.—PROGRESSIVE MUSCULAR ATROPHY (Soleus muscle, transverse section).
a, increased interstitial tissue; *b*, nearly normal muscle fibres; *c*, degenerated muscle fibres; *d*, atrophied muscle fibres; *e*, clusters of proliferated muscle nuclei.

Progressive muscular atrophy is apt to commence in the small muscles of the extremities, in many cases in the muscles of the ball of the thumb. It may commence in the muscles of the shoulder, the arms, or the back. It may have a continuous extension or it may jump single muscles or groups of muscles. Death may be induced by affection of the muscles of respiration or deglutition.

The causes of this lesion are in many cases unknown, and there is considerable lack of unanimity of opinion as to whether it is primarily a disease of the muscles or of the nervous system. In a considerable proportion of cases, the muscle lesion is associated with atrophy of the ganglion cells in the anterior cornua of the spinal cord and the development of connective tissue about them. In other cases, these changes in the cord may apparently be absent.

It is sometimes accompanied by atrophy of the nerves which are distributed to the muscles, and atrophy of the anterior roots has been described.

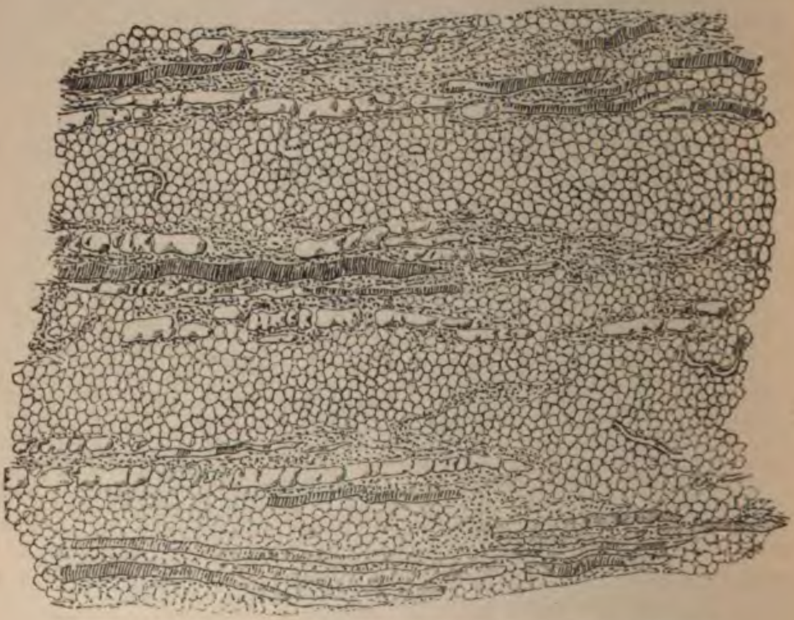

FIG. 208.—PSEUDO-HYPERTROPHY OF GASTROCNEMII MUSCLE (Fatty infiltration). This specimen was from the case mentioned below, accompanying multiple neuroma.

It is probable that there are several varieties of progressive muscular atrophy which our present knowledge does not enable us to clearly distinguish. Muscular atrophy in some cases follows overstraining of groups of muscles, or injuries, and may occur as one of the sequelæ of typhoid fever and diphtheria.

Atropia Musculorum lipomatosa (Pseudo-Hypertrophy of the Muscles).—In some cases, hand-in-hand with the production of new connective tissue in the muscles and the atrophy of the muscle fibres, or after these changes have made considerable progress, there occurs a development of fat-tissue between the fibres (Fig. 208) which may prevent any apparent diminution in the size of the muscles, or in some cases even give them a great increase in size. This condition is of most frequent occurrence in children, and is most apt to appear in the gastrocnemii muscles. In the upper extremities, the deltoid and triceps are most frequently involved. The lesion may be symmetrical, affecting similar muscles on both sides of the body, or it may be unilateral. Parts of muscle bellies may be affected.

The cause of this form of atrophy is not definitely known. Various lesions of the spinal cord have been described as occurring with it; but, in many cases at least, alterations of the nervous system cannot be detected. The writer has described a case [1] in which this lesion was marked in the gastrocnemii in connection with multiple false neuromata.[2]

Fatty Degeneration, with greater or less destruction of the muscles, may commence with a simple swelling and fine granulation of the fibres.

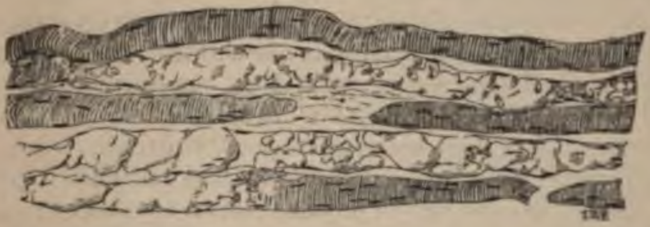

FIG. 209.—HYALINE DEGENERATION (SO-CALLED WAXY DEGENERATION) OF ABDOMINAL MUSCLE IN TYPHOID FEVER.

As the process goes on, smaller and larger fat-droplets appear in the contractile substance, which loses its striations and becomes friable, and may be entirely destroyed, leaving within the sarcolemma a mass of fatty detritus, which may finally be absorbed and disappear. This alteration may occur in acute parenchymatous myositis in connection with various forms of atrophy, in prolonged exhausting diseases, and in phosphorus poisoning.

Hyaline Degeneration.—Under a variety of conditions the muscle fibres undergo a peculiar series of changes, leading to their conversion into a translucent, highly refractile material, somewhat resembling amyloid, but not giving its micro-chemical reactions, and apparently more nearly allied to the material produced in the so-called hyaline degeneration. The lesion in the muscle which we are considering is commonly called *waxy degeneration,* from the peculiar appearance which the muscles present. When the lesion is far advanced and extensive, the muscles are brittle and have a grayish-yellow, translucent appearance. Microscopical examination of various stages of hyaline degeneration of muscle shows that the contractile substance of the fibres becomes at first swollen and granular and gradually converted into hyaline material, which may present the outlines of the swollen fibres, but is more frequently broken into larger and smaller shapeless clumps (Fig. 209), which may disintegrate and finally be absorbed. Hand-in-hand with these changes there usually occurs an increase in the interfibrillar connective tissue, and in certain cases there may be a proliferation of the muscle nuclei and a new formation of variously shaped cells within the sarcolemma, which leads to the regeneration of the fibres. As a result of the brittleness of the degenerated muscles, they are apt to rupture, and in this way hæmorrhage may occur.

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[1] *Prudden*, American Journal of Medical Sciences, July, 1880, p. 134.

[2] For bibliography of muscular atrophy, consult *Friedreich*, "Ueber progressive Muskelatrophie," etc., Berlin, 1873; also "Dictionnaire encyclopédique des Sciences Médicales," 2 ser. i., x.; or Eulenberg's "Real-Encyclopädie der gesammten Heilkunde," article by *Pick* on Muskelatrophie.

This form of degeneration may occur in progressive muscular atrophy, in variola, cerebro-spinal meningitis, trichinosis, in connection with inflammation, injuries, freezing, etc. It is, however, most marked and frequent in typhoid fever. In this disease the rectus abdominis and the adductors of the thigh are most frequently affected.

Experimental investigations have shown that under certain conditions very similar appearances may be produced in the muscles by post-mortem changes. It is not unlikely that a variety of changes are at present included under the name waxy or hyaline degeneration of the muscles.[1]

Hypertrophy of Muscle.—True hypertrophy of muscle as a pathological condition is rare, but it has been described in a few cases. It is usually confined to circumscribed groups of muscles. On microscopical examination, the diameter of the fibres is increased, sometimes considerably, though not uniformly. The transverse striation is unaltered, and the muscle nuclei are in some cases enlarged. The cause of the change is unknown.

TUMORS.

The tumors of the muscles usually develop in the connective tissue. *Fibroma, chondroma, lipoma, myxoma, sarcoma* may occur as primary tumors. *Carcinomata* and *sarcomata* may occur secondarily in the muscles as a result of local extension from adjacent parts. The muscle fibres are, as a rule, only secondarily affected by pressure, etc., in tumors of the muscles; but there exist observations which point to the possibility of a proliferation of the muscle nuclei and the new formation from them of cells which may take part in the growth of the tumor.

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[1] Consult *Zenker,* "Ueber die Veränderung der willkürlichen Muskeln in Typhus abdominalis," Leipzig, 1864; also *Weihl,* "Exp. Unters. ü. d. wachsartige Degeneration der quergestr. Muskeln." Virch. Arch., Bd. 61, p. 253, 1874.

PARASITES.

The *Trichina spiralis* is the most common parasite in the muscles. *Cysticercus cellulosæ* and *Echinococcus* occasionally occur.

PART IV.

THE LESIONS FOUND

IN

THE GENERAL DISEASES,

IN

POISONING,

AND IN

VIOLENT DEATHS.

TYPHOID FEVER.[1]

The lesions of typhoid fever are usually well marked and constant. They may conveniently be divided into two classes:

I. Those which are characteristic of the disease. To this class belong the changes in the lymphatic nodules (follicles) of the intestines, in the mesenteric lymph nodes (lymph glands), and in the spleen.

II. Those which are very frequently found with this fever and yet are not peculiar to it. To this class belong the changes in the parotid and pancreas, the degenerations in the liver, kidneys, and voluntary muscles, thrombosis of the blood-vessels, infarctions, diseases of the lungs, and suppuration of the connective tissue in various places.

I. *The Intestines.*—The lesions of the intestines consist in an inflammatory enlargement and subsequent degeneration of the solitary lymph nodules and Peyer's patches.

The process appears to begin with a catarrhal inflammation of the mucous membrane, accompanied or immediately followed by changes in the lymphatic follicles. The lesions in the lymphatic follicles begin early; they have been observed in persons who have died forty-seven hours after the commencement of the disease.

The increase in size of the agminated and solitary follicles may be rapid or gradual. The follicles may be only slightly enlarged, or may project so as to fill up the cavity of the intestine. The enlargement is usually more marked in the agminated than in the solitary follicles. Usually the whole of an agminated follicle will be enlarged, but sometimes only a part of it. If the enlargement is gradual, the different follicles which make up a Peyer's patch are enlarged, while the septa between them remain but little changed, and give the patch an uneven appearance.

The patches which are only moderately enlarged are of reddish or reddish-gray color, are soft and spongy, and their edges blend gradually with the adjoining mucous membrane. The patches which are more intensely affected are of gray or brownish color, of firm consistence, and rise abruptly from the surrounding mucous membrane, or even overhang it like a mushroom. The largest patches are sometimes more than three-eighths of an inch thick.

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[1] The most complete description of the lesions of typhoid fever is that given by *Hoffmann*, "Abdominal Typhus," 1869.

The enlargement and infiltration may spread from the patches to the surrounding mucous membrane, so that the patches appear very large; a number of them may become fused together, and there may be even an annular infiltration entirely around the lower end of the ileum.

The infiltration of the agminated follicles may also extend outward into the muscular coat, and even appear beneath and in the peritoneal coat as small, gray, rounded nodules. This condition is usually found only with a few patches in the lower end of the ileum; sometimes in the cæcum and appendix vermiformis. These little gray nodules usually correspond to diseased patches beneath them; sometimes they appear to excite an inflammation of the peritoneum, accompanied by the production of numbers of similar nodules all over that membrane. Hoffmann describes a case in which the inflammation extended to the pleura, with the production of similar nodules there.

The solitary follicles are affected in the same way as Peyer's patches. They may be hardly enlarged at all, or be quite prominent, or may be affected over a larger portion of the intestine than are the patches. Very rarely the solitary follicles are enlarged, while the patches are not at all or but slightly affected.

The inflammation and enlargement of the agminated and solitary follicles are followed by a healing process. The character of this process varies according to the intensity of the previous inflammation.

1. If the disease was mild and the enlargement of the follicles moderate, the enlargement gradually disappears and the follicles resume their normal appearance.

2. In moderate enlargements, the retrograde processes affect first the follicles, and leave the septa between them still swollen and prominent. This gives to the surface of a patch a reticulated appearance. After a time, however, the entire patch becomes flattened and uniform.

3. The solitary follicles or the separate follicles of a patch soften, break down, and their contents are discharged with some attendant hæmorrhage. This leaves a bluish-gray pigmentation in the situation of each follicle. This pigmentation may remain for years.

4. In more severe types of the disease, the enlargement of the follicles ends in ulceration. This takes place in two ways :

(*a*) The enlarged follicles soften, break down, and discharge into the intestine. In this way are formed small ulcers. These ulcers increase in size by the same softening process, which gradually attacks their edges, and in this way ulcers of large size may be formed. The ulcers may extend outward only to the peritoneal coat, or they may involve the peritoneal coat also and perforate.

(*b*) In the severest forms of the disease, considerable portions of the enlarged patches slough, are detached, and leave large ulcers with thick, overhanging edges. The slough may involve only the follicles, or it may involve also the muscular and peritoneal coats. These ulcers also may afterward increase in size, and several of them may be joined together.

If the patient recovers, the ulcers cicatrize, their edges become flattened, their floors are converted into connective tissue covered with cylindrical epithelium.

Both forms of ulceration sometimes end in perforation. This is effected by the extension of the ulcerative process through the peritoneal coat or by the rupture of the floor of the ulcer. Peritonitis and death are the usual result. In rare cases, however, the patient recovers and the perforation is closed by adhesions.

The minute changes which take place in the course of the intestinal lesion are as follows:

At first the blood-vessels around the follicles are dilated and congested, while the follicles are swollen and the epithelium falls off. Then the follicles increase largely in size from a growth of new cells. The new cells are, in part, similar to the lymphoid cells which normally compose the follicles; in part are large, rounded cells, some of which contain several nuclei. The production of new cells is not confined to the follicles, but extends also to the adjacent mucous membrane. In many cases also, little foci of the same cells are found in the muscular, subserous, and serous coats. This increased number of cells compresses the blood-vessels, and the parts become anæmic. Soon the cells degenerate, either by granular degeneration of individual cells or by gangrene of part of a follicle. In either case the degenerated portion is eliminated into the intestine, and leaves an ulcer of which the floor and edges are infiltrated with cells. After this the cell growth goes on, and the ulcer enlarges, or the cells are gradually replaced by connective tissue, and cicatrization follows.

The lesions we have described are found most frequently and most developed in the lower part of the ileum. They are not always, however, confined to this situation. Enlarged and ulcerated follicles may be found over the entire length of the ileum, and even in the jejunum. They may also extend downward and be found in the colon, even as far down as the rectum. The same changes may also take place in the appendix vermiformis.

Besides the regular typhoid lesions of the intestines which have been described, we occasionally meet with others of a more accidental character.

Gangrene of the intestinal wall sometimes occurs. It most frequently involves a portion of the wall corresponding to an ulcer, but may also affect other portions where no ulcer exists. The process may terminate in perforation or in healing.

Croupous Inflammation may attack the mucous membrane of either the large or small. intestine. The mucous membrane between the typhoid ulcers is covered and infiltrated with an exudation of fibrin and pus.

Peritonitis of a mild type is a frequent accompaniment of the intestinal lesions. It appears to have but little influence on the course of the disease.

Severe peritonitis is usually due to perforation, less frequently to ulcers which reach the serous coat but do not perforate. When there is infiltration of the serous coat with the typhoid new growth, the peritonitis may be accompanied by a production of little gray nodules of the same character throughout the peritoneum.

Infarctions of the spleen. inflammation of the ovaries, and perforation of the gall bladder are sometimes the cause of peritonitis.

Hæmorrhage from the intestines is merely due to the inflammatory swelling and congestion of the mucous membrane, and is slight; or it is due to the ulceration of the follicles and destruction of the blood-vessels, and is then often profuse.

Mesenteric Lymph Nodes.—The mesenteric nodes undergo the same changes as the follicles of the intestines, and are usually affected in a degree corresponding to the intensity of the intestinal lesion.

The nodes are at first congested and succulent; then there is a production of lymphoid cells and large cells, as in the intestinal follicles (Fig. 152), and the node becomes enlarged. When the enlargement has reached its full size, the congestion diminishes and the cells begin to degenerate. The degeneration may take place slowly, and then the node gradually returns to its normal condition; or more rapidly, and then little foci of softened purulent matter are formed. If the patient recovers, the small foci are absorbed, leaving a fibrous cicatrix; the larger foci become dry, cheesy, and inclosed in a fibrous capsule. The inflammation of the nodes may produce a local or general peritonitis.

The Spleen.—In nearly every case of typhoid fever, the spleen is enlarged. This enlargement begins soon after the commencement of the disease, increases rapidly until the third week, remains stationary for a few days, and then diminishes. The organ is congested, of dark-red color, and of firm consistence while it is increasing in size. After it has reached its maximum size, its consistence becomes soft, and there is a considerable deposit of brown pigment. The enlargement appears to be due to the congestion and to an increase of the normal elements of the spleen.

In rare cases, the softened spleen is ruptured, with an extravasation of blood into the peritoneal cavity.

There may be infarctions of the spleen, which sometimes soften and cause peritonitis.

II. The second class of lesions comprises those which are frequently found with typhoid fever, but are not peculiar to it.

The Mouth.—A number of changes are found about this region. The follicles at the root of the tongue and the tonsils may be enlarged; the muscles of the tongue may undergo waxy and granular degeneration; gangrenous ulcers may attack the floor and sides of the mouth and destroy large areas of tissue.

The Pharynx may be the seat of catarrhal or croupous inflammation, producing superficial and deep ulcers.

The Parotid is, in a moderate number of cases, the seat of an inflammation which tends to suppuration. In this process, both the glandular acini and the connective tissue between them are involved. Which of the two has the larger share in the process is still in dispute.

A slight enlargement and induration of the parotid and submaxillary glands is said by Hoffmann to be a frequent lesion, and to depend on increase of the gland cells and dilatation of the follicles with their secretion.

The Pancreas undergoes changes similar to those in the salivary glands. It becomes at first swollen and red, then hard and grayish, then yellow. The vessels are at first congested, afterward there is increase of the gland cells, and lastly degeneration.

The Liver may preserve its normal character or may present changes.

In many cases, the organ will be found soft and flabby. Minute examination then shows that the liver cells have undergone degeneration. They are filled with fine granules and small fat-globules, and the degeneration may go on so far that the outlines of the hepatic cells are lost and nothing but a mass of granules can be seen.

Less frequently we find in the liver very small, soft grayish nodules resembling those found in the peritoneum. They are situated along the course of the small veins, and there is, at the same time, a diffuse infiltration of lymphoid cells along the small veins. The nodules consist of lymphoid cells; they are often too small to be distinguished with the naked eye.

The Heart.—In a considerable number of cases, the muscular tissue of the heart is altered. The heart feels soft and flabby; it is of grayish or brownish color; the muscular fibres are infiltrated with fine granules, and sometimes with brown pigment. Or the heart is firm, but friable and easily torn, its cut surface glistens, and its muscular fibres are in the condition of hyaline degeneration.

Thrombi in the cavities of the heart and vegetations on the valves are sometimes found. Detached fragments of these may be lodged as emboli in the different arteries.

The Arteries.[1]—There may be an acute inflammation of the arteries, especially at the commencement of convalescence. There are two varieties: an obliterating and a parietal arteritis. In the obliterating arteritis there is infiltration of all the coats of the artery, with roughening of the intima and the formation of a thrombus within the vessel, and this is followed by dry gangrene of the parts supplied by the artery. In the parietal variety the wall of the artery is infiltrated with cells, but the intima is not roughened and no thrombus is formed.

The Veins.—Thrombosis of the larger veins, especially of the femoral vein in the third and fourth weeks of the disease, is not uncommon.

The Larynx is very frequently the seat of catarrhal inflammation, with or without superficial erosions. Less frequently there is croupous inflammation, followed in some cases by destructive ulceration.

The Lungs.—Catarrhal inflammation of the large bronchi is very common. Broncho-pneumonia occurs in two forms. There may be a severe inflammation of most of the bronchi of both lungs, with cellular infiltration of the walls of the bronchi and zones of peribronchitic pneumonia; or there is an intense general bronchitis, with lobules of the lung corresponding to obstructed bronchi, either collapsed or inflamed, or both.

From the long-continued recumbent position of the patients, the posterior portions of the lungs become congested, dense, and unaërated. Sometimes, in addition to this, irregular portions of the lungs become hepatized.

Less frequently there is regular lobar pneumonia.

There may be infarctions in the lungs.

Gangrene of the lungs is occasionally found, either associated with lobular pneumonia or with infarctions, or as an independent condition.

The Kidneys very frequently present the lesions of parenchymatous nephritis. They may contain infarctions.

The Ovaries.—Hæmorrhage and gangrenous inflammation have been observed in rare cases.

The Testicles.—Orchitis has been described by Ollivier.[2] It generally is developed during convalescence; it is unilateral; it usually affects the testicle alone, less frequently the epididymis; it terminates in suppuration in nearly one-fourth of the cases.

The Brain.—Acute meningitis, thrombosis of the venous sinuses, and obliterating endarteritis of the cerebral arteries occasionally are observed.

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[1] *Barié*, Rev. de Méd., Jan., Feb., 1884. *Keen*, "Toner Lectures on the Surgical Complications of the Continued Fevers," 1877.

[2] Rev. de Méd., Nov. and Dec., 1883.

The Voluntary Muscles, especially the abdominal muscles, the adductors of the thigh, the pectoral muscles, the muscles of the diaphragm and of the tongue, frequently undergo the hyaline degenerative changes described under muscle lesions (Fig. 209).

The Skin.—Gangrenous inflammation of the skin frequently occurs in the form of bed sores, affecting especially the skin over the sacrum and trochanters, where it is subjected to the constant pressure of the bed.

There may be suppurative inflammation of the connective tissue in any part of the body. Perhaps the most important of these local suppurations is that which produces retro-pharyngeal abscesses.

The Bacillus of Typhoid Fever.—The presence of a bacillus in various parts of the body in typhoid fever, in a considerable proportion of the cases examined, has been well established by a large number of observers. This bacillus does not occur in the body, so far as is known, except in connection with this disease.

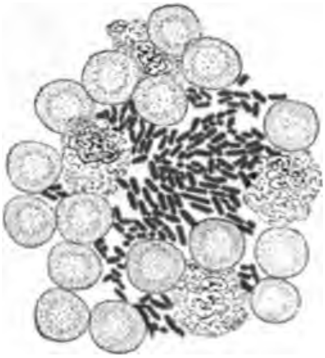

FIG. 210.—CLUSTER OF TYPHOID BACILLI IN THE SPLEEN.

In the early stages of the disease, the bacillus may be found in the lymphatic structures of the intestines, and in the mesenteric lymph nodes and the spleen. It may be present in the kidney, liver, lungs, and in the blood, and is often found in enormous numbers in the intestinal contents. In the viscera it is apt to occur in larger and smaller masses or clusters (see Fig. 210).

The typhoid bacillus is usually about three times as long as broad (Fig. 211), being about one-third as long as the diameter of a red blood-cell. It is rounded at the ends, and frequently contains rounded structures which have been regarded as spores, but which further researches have led us to believe are not spores but vacuoles.

The typhoid bacillus can be readily cultivated on the ordinary culture media at room temperatures. It forms delicate, bluish white, sinuousedged spreading colonies on the surface of nutrient gelatin, which it does not fluidify. Several other bacteria grow in a similar way on gelatin, but the mode of growth of the typhoid bacillus on boiled potatoes is its most marked culture characteristic; for, unlike any other known species, it forms on the surface of the potato an invisible pellicle. If, however, the potato be made slightly alkaline, the surface growth becomes evident. In cultures, the typhoid bacilli often cling together end to end, forming long, thread-like structures (Fig. 211). The bacilli in fluids are actively mobile.

Inoculations of the typhoid bacillus into animals, while not producing a disease in all respects like that in the human subject, may cause death with symptoms and lesions as closely resembling those in man as we are often able to produce in animal experimentation. Altogether the evidence that typhoid fever in man is produced by the typhoid bacillus, and by this alone, is so strong as practically to amount to a demonstration.

It is probable that the symptoms and lesions of typhoid fever are largely due to the absorption of a ptomaïne which is produced as the result of the life processes of the bacteria at the point of their greatest accumulation and activity, namely, in the intestinal canal.

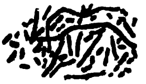

FIG. 211.—TYPHOID BACILLI FROM GELATIN CULTURE.

Typhoid fever appears in a large proportion of cases to be communicated by means of drinking water which has been polluted with the excretions of persons suffering from the disease.

The bacilli are capable of living for considerable periods in water as well as in ice. They have been repeatedly found by biological examinations in polluted drinking water to which external evidence has pointed as the source of a typhoid epidemic.

METHODS OF STAINING THE TYPHOID BACILLUS.

The bacilli, when taken from cultures, stain readily with the ordinary aniline dyes, such as fuchsin and gentian violet (see page 96).

In sections of the organs they do not take the stain so readily. They are decolorized by Gram's method.

One of the most satisfactory solutions for this purpose is that of Ziehl, which is made as follows:

Filtered Saturated Aqueous Solution of Carbolic Acid...	90
Saturated Alcoholic Solution of Fuchsin..............	10

The sections are soaked for half an hour in this solution and then decolorized by alcohol, cleared in oil of cedar, and mounted in balsam. The decolorization should be done carefully, the section being examined from time to time as it proceeds, so as to avoid the removal of too much color. The nuclei should remain faintly colored, but not so much so as to conceal the clusters of more deeply stained bacilli.[1]

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[1] For literature of typhoid bacillus, consult a résumé by *Seitz*, Centralblatt für Bakteriologie u. Parasitenkunde, Bd. 2, No. 23, 1887.

TYPHUS FEVER.

This disease has not, so far as we know, any characteristic lesion; but yet after death we may find a number of morbid conditions, such as are common to the infectious diseases.

The entire body has a tendency to rapid putrefaction.

The blood is often darker and more fluid than in other diseases.

The voluntary muscles may undergo waxy and granular degeneration.

The brain and its membranes may be congested.

The mucous membrane of the pharynx and larynx may be the seat of catarrhal or croupous inflammation.

In the lungs there may be bronchitis, broncho-pneumonia, or hypostatic congestion.

The walls of the heart may be soft and flabby.

The agminated glands of the ileum, and the mesenteric nodes, may be a little swollen.

The spleen is often large and soft.

The kidneys are frequently the seat of parenchymatous nephritis.

The nature of the infective agent in typhus is unknown.

RELAPSING FEVER.

Synonyms.—Typhus recurrens; Famine fever; Spirillum fever.

The Skin may be jaundiced; it may be mottled by extravasations of blood.

The Brain and *Spinal Cord* are unchanged.

The Pharynx and *Larynx* may be the seat of catarrhal or croupous inflammation.

The Lungs.—There may be bronchitis, broncho-pneumonia, lobar pneumonia, hypostatic congestion, and pleurisy.

The Heart is often soft and flabby, with degeneration of its muscular fibres. There may be ecchymoses in the pericardium.

The Stomach and *Small Intestine* may be congested; there may be ecchymoses in the mucous membrane; there may be catarrhal inflammation.

The Colon may be the seat of catarrhal or croupous inflammation.

The Mesenteric Nodes may be swollen.

The Liver is often enlarged and the hepatic cells are swollen and granular.

The Spleen is large and soft, like the spleen of typhoid fever. The change in its consistence is so marked that the spleen may rupture spontaneously during life. The spleen may also contain infarctions of different sizes; some are red, some yellow, some necrotic. Those which are necrotic may give rise to a local or general peritonitis.

The Kidneys show the lesions of parenchymatous nephritis.

The Bones.—Degenerative changes in the medulla of the bones have been described by Ponfick.[1]

Bacteria.—In the blood of all parts of the body during the febrile attacks may be found, in very large numbers, a long, slender spirillum called *Spirochæte Obermeieri.* It disappears from the blood during the afebrile intervals. The organism is from sixteen to four *μ* in length, and performs rapid, undulating movements (Fig. 212). The inoculation of monkeys with the blood of relapsing-fever patients which contains the bacteria induces a similar disease. *Pure cultures* have not as yet been made of these bacteria, but for the reasons indicated, and since the organism has never been found except in connection with the disease, there is every reason for believing that the Spirochæte Obermeieri is the cause of relapsing fever.

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[1] Virch. Arch., Bd. 60, p. 153.

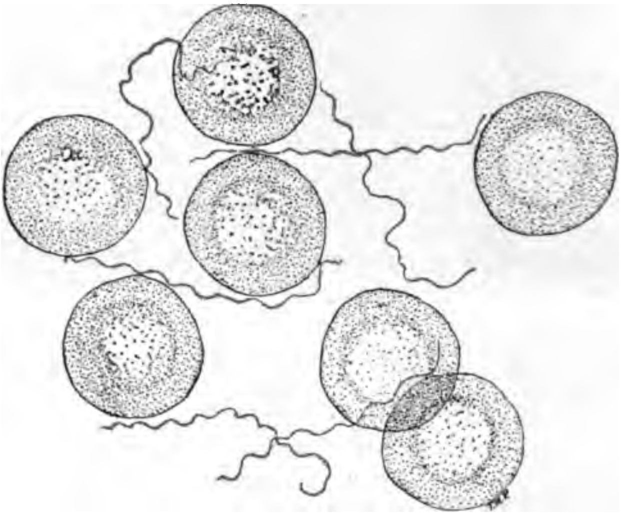

FIG. 212.—SPIROCHÆTE OBERMEIERI IN THE BLOOD IN A CASE OF RELAPSING FEVER.

ACUTE CEREBRO-SPINAL MENINGITIS.

This is an acute infectious disease, characterized by an exudative inflammation of the pia mater of the brain and spinal cord. The degree of the lesion in the meninges varies greatly, depending upon the period at which death occurs. In some cases, when death occurs early in the disease, there may be to the naked eye no evident change in the membranes, or a moderate serous infiltration. In these cases, the microscope may reveal a moderate degree of extravasation of leucocytes in the vicinity of the vessels. In the well-marked cases, the pia mater of the brain and cord is more or less densely infiltrated with serum, fibrin, and pus. This may occur over the convexity and base of the brain, and is frequently most marked in the latter situation. In the cord, the infiltration may occur over the anterior and posterior surfaces; but in many cases, probably owing to the recumbent position of the patient, it is most marked on the posterior surface. The ventricles of the brain and the central canal of the cord may contain turbid serum mingled with pus-cells, and sometimes blood-cells. The membranes and underlying nerve tissue may be hyperæmic and the seat of capillary hæmorrhages. Rarely a small amount of pus and fibrin may be found between the pia and dura mater.

In protracted cases, the ventricles may be dilated with serum, and the exudation in the meninges may become fatty or dense and cheesy.

While the above are the characteristic lesions of this disease, there are a number of secondary changes in different parts of the body which are not constant, but which occur with sufficient frequency to render their mention necessary. There may be subserous punctate hæmorrhages in the endocardium; petechiæ in the skin; hyaline and granular degeneration in the voluntary striated muscle; occasional multiple abscesses in various parts of the body; suppurative inflammation of the joints; parenchymatous degeneration of the heart, liver, and kidneys; and swelling of the gastro-intestinal lymphatic apparatus.

Cerebro-spinal meningitis may occur by itself or in connection with some other acute infectious disease, such as acute lobar pneumonia, mycotic ulcerative endocarditis, pyæmia, multiple suppurative arthritis, otitis media, puerperal fever, typhoid fever, etc.

The lesions are essentially the same in epidemic and in sporadic cases of acute cerebro-spinal meningitis, and there is much reason to believe that in both modes of occurrence the disease is caused by bacteria. But studies of the epidemic form of the disease have not yet been made by the new methods, so that we can say nothing definite about its causation.

On the other hand, numerous careful studies have been made on the bacteria occurring at the seat of lesion in sporadic cases occurring both with and without complicating lesions in other parts of the body.

The *Streptococcus pyogenes* has been demonstrated in a few cases, occurring in connection with suppurative inflammations elsewhere.

The *Pneumococcus of Fraenkel* (see page 238) has been found in several cases, and in some of these without any lung lesion. Weichselbaum has described the occurrence in several cases of a diplococcus not known to occur elsewhere, which was found largely confined to the pus cells, and which he called *Diplococcus intracellularis meningitidis.*

Animal experiments with this as well as the pneumococcus would indicate that they stand in a causative relation to the disease. Some other scattering forms of bacteria have been described, but not with sufficient frequency and definitiveness to enable us to judge of their significance.

It seems probable, therefore, from what we know at present, that several forms of bacteria are capable of causing acute cerebro-spinal meningitis. Which is the most frequent and important it remains for further researches to show.[1]

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[1] For literature and further details consult *Weichselbaum,* Fortschritte der Medicin, September 15th, 1887; *Goldschmidt,* Centralblatt für Bakteriologie, Bd. 2, No. 22; *Neumann and Schaefer,* Virchow's Archiv, Bd. 109, p. 477.

DIPHTHERIA.

Diphtheria is an acute infectious disease, usually characterized by a croupous inflammation on some of the mucous membranes or on the surface of wounds.

The mucous membranes which are the most frequently affected in diphtheria are those of the tonsils, pharynx, soft palate, nares, larynx, and trachea; less frequently those of the mouth, gums, œsophagus, and stomach.

The local inflammation may present various phases which represent clinical types of the disease. Thus there may be a simple redness of the affected surfaces which leaves no trace after death, or a catarrhal inflammation. On the other hand, in the more marked forms of the lesion there may be a fibrous exudate which infiltrates the mucous membrane, or, intermingled with pus cells, epithelial cells, red blood-cells, bacteria, and granular material, forms a thick or thin pellicle on the affected surfaces. This pellicle may undergo coagulation necrosis, and hand-in-hand with this there may be superficial or deep coagulation necrosis of the mucous membrane.

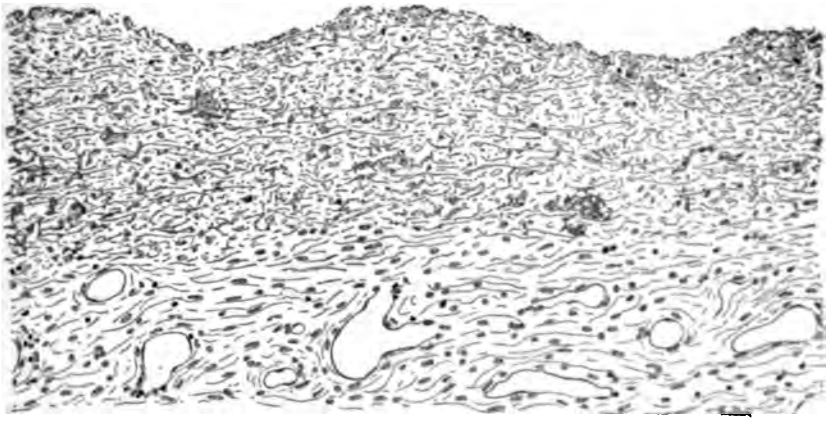

FIG 213.—DIPHTHERIA OF THE TRACHEA.

Section through the pseudo-membrane and underlying tissue, showing large numbers of streptococci.

Bacteria of various forms are commonly present in the false membrane, and some of the forms may penetrate deeply into the underlying tissue.

The false membrane in diphtheria is thus formed by a combination of inflammation and necrosis, the extent of the necrosis and the amount of inflammatory products varying in the different cases.

The pseudo-membrane may disintegrate or exfoliate, with or without loss of tissue in the underlying mucous membrane. Phlegmon, abscess, and œdema are liable to occur as local complications. Adjacent and distant lymph nodes are apt to be swollen, and the liver, kidneys, and salivary glands to present parenchymatous degeneration.

Catarrhal bronchitis, and broncho-pneumonia or lobular pneumonia, frequently complicate diphtheritic lesions of the upper air passages and fauces.

Prolonged series of studies have shown that diphtheria is caused by bacteria, which apparently induce the croupous inflammation by their local growth, and the systemic effects by the absorption of a ptomaïne produced by the germs at the seat of local lesion.

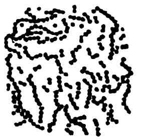

FIG. 214.—STREPTOCOCCUS DIPHTHERIÆ. From a beef-tea culture.

It is not unlikely that more than one species of bacteria is capable of inducing that series of symptoms or lesions which we call diphtheria.

Loeffler and others have isolated a slender bacillus from the pseudo-membrane in several cases, which they are disposed to believe to be the cause of the disease in the cases which they studied in Germany.

On the other hand, bacterial studies on cases of diphtheria occurring in New York have failed to reveal the presence of Loeffler's bacillus, but have shown the very constant presence in and about the local lesion (Fig. 213) of a streptococcus which appears to be identical with the streptococcus of suppuration and of erysipelas, and which there is much reason for believing is the cause of the disease (Fig. 214). These bacteria, which have been temporarily called *Streptococcus Diphtheriæ*, are not found in any considerable numbers in the viscera.[1]

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[1] For a résumé of earlier bacterial investigations on diphtheria, and the results of recent studies on cases occurring in New York, see "Studies on the Etiology of Diphtheria" (*Prudden*), Am. Jour. Med. Sc., April and May, 1889.

ASIATIC CHOLERA.

In some cases of cholera there are no marked changes to be found after death.

If death occurs during the invasion of the disease or in the stage of collapse, in the more marked cases the appearances are as follows:

The bodies remain warm for some time, and the temperature may rise for a short time after death. The rigor mortis begins soon and lasts for an unusually long time. The muscles sometimes exhibit a peculiar spasmodic twitching before the rigor mortis sets in, especially the muscles of the hand and arm.

The Skin is of a dusky gray color, the lips, eyelids, fingers, and toes of a livid purple. The ends of the fingers are shrivelled, the cheeks and eyes are fallen in.

The Brain.—The sinuses of the dura mater are filled with dark, thick blood. The pia mater may be normal, or œdematous, or ecchymosed, or infiltrated with fibrin. The brain is usually normal, but may be dry and firmer than usual.

The Lungs are retracted and anæmic, the pleura may be dry or coated with fibrin.

The Heart is normal.

The Peritoneum may be dry or coated with a layer of fibrin.

The Stomach is usually unchanged, but may be the seat of catarrhal inflammation.

The Small Intestine.—There may be ecchymoses in the mucous membrane; the mucous membrane may be soft and œdematous; there may be general congestion, or the congestion may be confined to the peripheries of the solitary and agminated glands, and these glands may be swollen; or there may be croupous inflammation and superficial necrosis. All these changes are regularly most marked at the lower end of the small intestine. There is apt to be post-mortem desquamation of the epithelium. The characteristic rice-water fluid may be found in the intestines after death, or instead of this a dark-colored, bloody fluid.

The Large Intestine is usually normal, but in some epidemics croupous inflammation occurs in a considerable number of cases.

The Liver and *Spleen* undergo but few changes; they may be anæmic and flabby.

The Kidneys are often increased in size, with white and thickened cortex and congested pyramids. The epithelium of the cortex tubes contains coarse granules and fat-globules. The tubes contain cast matter and broken-down epithelium. These changes may be looked upon as being simply of a degenerative character or as the results of a parenchymatous nephritis.

The Uterus and *Ovaries* may be congested and contain extravasated blood.

If the patient does not die until the stage of reaction, the body does not present the same collapsed appearance, and there are often inflammatory changes in different parts of the body, especially in the larynx, the lungs, the stomach, and the intestines.

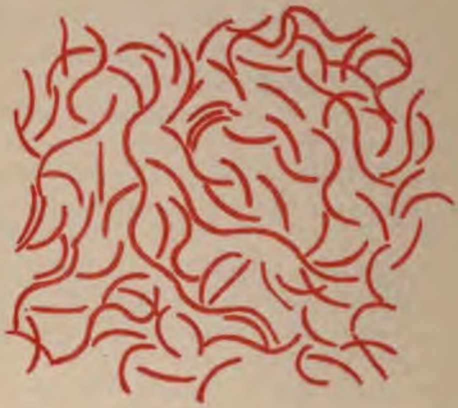

FIG. 215.—SPIRILLUM CHOLERÆ ASIATICÆ. From a beef-tea culture.

According to recent researches by Koch, which have been abundantly confirmed by others, there are constantly present in the small intestines of cholera patients, during the early and active stages of the disease, characteristic curved bacilli which are not known to occur in the body under any other conditions, and which have been proved to cause the disease. These bacilli are from 0.8 to 2.0 μ long, and are sometimes slightly, sometimes considerably curved (see Fig. 215). When growing, the individual bacilli are apt to cling together by their ends, forming S-shaped figures or spirils of considerable length. They are therefore often called *Spirillum choleræ asiaticæ*. From the curved shape of the individuals they are also often called "comma bacilli." They may be present in moderate numbers in and beneath the mucous membrane of the intestine, and in very large numbers in the intestinal contents and in the dejections in the acute forms and early stages of the disease.

In the process of their growth and multiplication in the intestinal canal, they apparently produce a poisonous ptomaïne, the local action and absorption of which into the body fluids produce the symptoms and lesions of the disease. The systemic effects appear to be in the nature of a septic intoxication. The cholera bacillus may retain its vitality for a considerable period in water, and on moist substances, such as damp linen, earth, and vegetables, may increase in numbers with great rapidity.

A temperature of from 30°–40° C. is most favorable for their growth. At about 16° C. their proliferative activities cease, but they are not killed by −10° C. They are readily killed by drying, and the presence of acids is very inimical to their growth.

There is not sufficient evidence that they form spores, and their period of life is short.

The cholera bacillus is readily cultivated on artificial culture media, such as gelatin, agar, milk, beef-tea, potatoes, etc. In fluids it is capable of performing active movements.

While they may be readily stained by the ordinary methods when present in the dejecta, their morphological characters are not absolutely distinctive, since several forms of curved bacilli belonging to the same group and closely resembling them have, under varying conditions, been found in the dejecta and in the mouth.

It is often of the highest importance to determine, at the earliest possible movement, whether or not a suspected case be one of Asiatic cholera or some other form of acute intestinal disorder, so that the proper measures may be instituted to prevent the spread of the disease. The characters which are developed in the cultures of the cholera bacillus enable an expert biologist to distinguish this organism from all other known forms.

Gelatin plate cultures are made in the usual way (see page 101) from the intestinal contents, very small portions being taken. A considerable number of plates should be made of varying dilutions and kept at about 21° C. (70° F.). Usually after about twenty-four hours, the colonies of the cholera germ appear as small, rounded, grayish, granular bodies with rough edges. As the colonies grow, they become more coarsely granular, and after a time look as if composed of strongly refractile granules, like particles of pounded glass. The gelatin becomes fluidified around the growing colony, which sinks into the fluid-filled pit thus formed. In gelatin tubes inoculated in the usual way with the cholera bacillus, the gelatin is fluidified along the puncture line, but most rapidly at the surface, where a small air bubble usually lies at the top of the fluidified pit.

On potatoes the cholera bacillus grows but slightly at ordinary temperatures, but in the thermostat, at the temperature of the body, it grows rapidly, forming a light-grayish or brownish pellicle.

By thus taking together the morphological and biological characters, it is possible usually on the second or third day to determine whether the intestinal contents of a suspected case does or does not contain the bacillus of Asiatic cholera.

The cholera bacillus, both in the dejecta and in pure cultures, is readily stained by the ordinary aniline dyes. The results of the introduction of considerable quantities of cholera bacillus cultures into the gastro-intestinal canal of animals have abundantly proven the causative relation of the bacillus to the disease.[1]

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[1] For the details of his researches on Asiatic cholera, see *Koch's* report, "Arbeiten a. d. kaiserlichen Gesundheitsamte," Bd. 3, 1887.

YELLOW FEVER.

The Skin is of a yellow color from the presence of bile pigment, and may be mottled by ecchymoses.

The Heart is of a pale or brownish-yellow color. Its muscular fibres are the seat of fatty degeneration.

The Lungs are congested.

The Stomach often contains the characteristic black fluid which is vomited during life. Its mucous membrane is congested, softened, and sometimes eroded.

The Intestines are dark-colored, often distended with gas, and sometimes contain blood.

The Liver in the earlier stages of the disease may be intensely congested. More frequently it contains but little blood, is of a light-yellow color, and the hepatic cells are infiltrated with coarse granules and fat-globules. The gall bladder is contracted and contains but little bile.

The Spleen shows no marked changes.

The Kidneys present the lesions of the intense form of parenchymatous nephritis.

While its mode of occurrence and the character of its symptoms and lesions afford a strong presumption that yellow fever is an acute infectious disease, none of the various studies which have been made upon its etiology have as yet revealed the presence of any micro-organism to the action of which it can be fairly attributed.

TUBERCULOSIS.

Persons are said to have tuberculosis when there is going on, in some part of their body, an inflammation accompanied with the growth of tubercle bacilli.

It is also possible that persons may have tubercle bacilli in the blood and tissues without any inflammation, but of this we have no actual knowledge.

In the experimental production of tuberculosis in animals, the inoculation of pure cultures of the bacillus is followed by inflammatory changes at the point of inoculation, and then the infection of other parts of the body through the blood-vessels or the lymphatics. In the tissues thus infected inflammatory changes take place. It seems, therefore, that the tubercle bacilli act as the direct cause of the inflammation. It has been found, however, that the spread of the bacilli from the point of inoculation is very decidedly influenced by the environment and mode of life of the animal, and that some kinds of animals are much more susceptible to the infection than are others.

In human beings, cases of direct local inoculation have been reported, but they are very rare.

There seems to be no doubt that the bacilli can be introduced into the alimentary canal by infected milk and meat. It is still uncertain, however, how often such a mode of infection really takes place.

The ordinary way for tubercle bacilli to be introduced into the human body seems to be by the air inspired into the lungs.

Tuberculosis occurs most commonly in the form of a tubercular inflammation affecting some one part of the body—"localized tuberculosis." Such a localized tuberculosis may retain throughout the characters of a local inflammation; it may be accompanied by the clinical evidences of systemic infection; it may give rise to the successive development of tubercular inflammation in other parts of the body; it may give rise to a sudden development of tubercular inflammations in many parts of the body at the same time.

It seems possible that such localized tubercular inflammations may be due to the presence of the tubercle bacillus acting as a local irritant.

More frequently, however, it is necessary that a traumatism, or some other cause of inflammation, should set up an inflammation to which the tubercular character is given by the growth of the bacillus.

The development of secondary tubercular inflammations may be due either to simple infection with the bacilli or to the action of fresh causes of inflammation.

If there is a sudden formation of miliary tubercles in many parts of the body at the same time, the patient is said to have "general miliary tuberculosis."

Such a general infection may be caused by the diffusion through the body of bacilli derived from a local tuberculosis, such as tubercular phlebitis or arteritis, or from the breaking into a vessel of a tubercular lymph gland, or by the inhalation into the lungs of large numbers of bacilli.

The hereditary constitution, the mode of life, the climate, all have a decided effect in rendering each individual more or less liable to tubercular infection.

The method by which human beings transmit tuberculosis seems to be largely by means of the dried sputa.

The forms of inflammation which are excited by or accompany the tubercle bacillus are the exudative and the productive.

The inflammations run an acute, subacute, or chronic course.

The lesions which we regularly find are:

1. Miliary tubercles.
2. Diffuse inflammation of various kinds, with cheesy degeneration of the inflammatory products.
3. The ordinary products of inflammation—pus, fibrin, serum, epithelium, granulation tissue, and connective tissue.

Associated with all these lesions we find the tubercle bacilli.

1. *Miliary Tubercles.*—These are small nodules, of irregularly spheroidal shape, the smallest hardly visible to the naked eye, the largest as large as a pea. The smaller ones are gray and semi-transparent; the larger are opaque, whitish or yellow, especially at their centres.

Miliary tubercles do not all have the same structure.

Some are composed of amorphous granular matter, of degenerated lung tissue, and of epithelial cells and pus.

Some are composed of a tissue resembling granulation tissue.

Some are composed of tubercle tissue, alone or associated with other inflammatory products (Figs. 44 and 128).

The term "tubercle tissue" is employed to designate an inflammatory product which somewhat resembles granulation tissue. It is composed of a basement substance and of cells. The basement substance is delicate and finely granular, and contains round and oval nuclei. This basement substance has a reticulated arrangement, and in the spaces of the reticulum are polyhedral nucleated cells. There may also be present the large nucleated bodies called giant cells. These giant cells, although apparently all formed in the same way by the fusion of a number of smaller cells, yet do not always present exactly the same appearance. Some of them seem to form part of, and to be continuous with, the basement substance; others are separated from the basement substance, and look like large cells contained in the meshes of the basement substance. In some tubercle tissue the basement substance, in others the polyhedral cells, in others the giant cells are predominant (Figs. 42 and 43).

Such tubercle tissue is arranged in the form of small spheroidal bodies—tubercle granula—and of a diffuse tissue. So that a "tubercular" miliary tubercle is composed of one or more tubercle granula and of diffuse tubercle, to which may be added other inflammatory products.

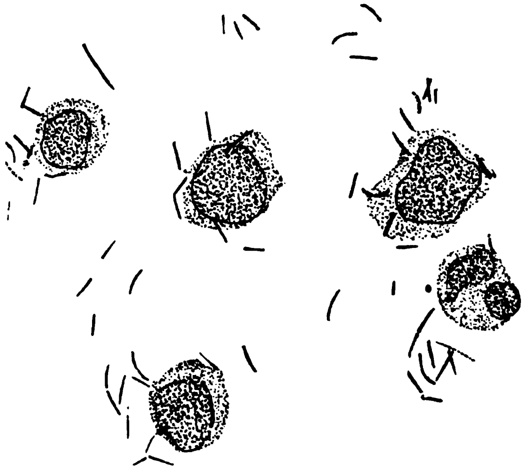

FIG. 216.—TUBERCLE BACILLI, WITH PUS CELLS, IN SPUTUM. The bacilli stained with fuchsin.

All miliary tubercles may undergo cheesy degeneration, or, more rarely, be changed into connective tissue.

The miliary tubercles formed of amorphous granular matter or of indifferent round-celled tissue are most common in acute general tuberculosis. The miliary tubercles formed of well-defined tubercle tissue are most completely developed in localized tuberculosis.

Tubercle tissue infiltrates and replaces connective tissue; it fills cavities, and it projects from free surfaces. It contains but very few blood-vessels, and when it infiltrates a tissue the vessels of that tissue become obliterated. There is often associated with it a proliferation of the endothelial cells of the arteries or an obliterating endarteritis.

2. *Diffuse Inflammation with Cheesy Degeneration of the Inflammatory Products.*—This form of lesion is found in the large tubercular masses in the brain, in the mucous membrane of the bronchi, in the large flat tumors of the serous membranes, and in the diffuse, cheesy infiltration of the kidneys, the ureters, bladder, prostate, testicle, and uterus. The constant feature of the lesion is the development of an indifferent round-celled tissue, which rapidly undergoes cheesy degeneration. Imbedded in this tissue there may be tubercle granula. When the lesion is far advanced, the degenerative changes may involve all the inflammatory products, so that we find no formed elements, nothing but a diffuse, caseous mass. In the earlier stages of the lesion, the indifferent round-celled tissue and the tubercle granula are readily demonstrated.

3. *Pus, fibrin, serum, epithelial cells, connective tissue, and indifferent tissue* are all found in varying quantities, either separately or together, in those parts of the body where the tubercular lesions have been developed.

The Tubercle Bacilli are slender, filamentous bacteria (Fig. 216), varying in length from one-fourth to one-half the diameter of a red blood-cell. They may contain spores, which give them a beaded appearance, and are sometimes slightly curved. It is possible, but very difficult, to see them when they have not been stained. Morphologically they very closely resemble the bacilli of leprosy and some other forms of filamentous bacteria. They differ from most similarly formed bacilli in the tenacity with which they retain the color after staining, although the bacilli of leprosy have this same property. It is, however, the effect of the living bacilli upon the organism, when introduced into it under favorable conditions, which furnishes the only absolute proof of their identity.

The tubercle bacilli are present in the characteristic lesions of almost all cases of local and general tuberculosis. But there are apparently exceptional cases of typical tuberculosis in which the most careful examination fails to reveal the bacilli. This apparent absence of the bacilli is probably due either to their disappearance as the process grows older, or to some unknown changes which interfere with the ordinary staining procedures.

The number of bacilli which are present in the lesions is subject to great variations. They are most abundant in the walls and contents of phthisical cavities, and in tubercle tissue which is undergoing cheesy degeneration and disintegration. In these situations they may be found in myriads, forming sometimes a large part of the disintegrated mass. They are found in cells and scattered among them. Sometimes they are present in considerable numbers in the giant cells of miliary tubercles. In the dense, firm, chronic miliary tubercles they are not commonly present. In the acute general tuberculosis of children they are often present in enormous numbers, particularly in the lungs. They may be found in tubercular inflammation in any part of the body, and have been seen in the blood. The bacilli are almost constantly found in the sputa of phthisical patients, and their presence sometimes affords valuable diagnostic aid in early stages or obscure forms of the disease.

METHODS OF STAINING THE TUBERCLE BACILLI.

In Fluids.—The most reliable and useful of the numerous methods which are employed for staining the tubercle bacilli is *Ehrlich's* or some modification of this. For the examination of fluids, such as sputum, etc., the material should be spread in a thin layer on a cover-glass, dried in the air, and then passed thrice through the flame (see page 96). The staining fluid is prepared by making (1) a saturated solution of anilin oil in distilled water; this is done by adding about five per cent of the oil, and shaking vigorously and filtering through a moistened filter. To this clear filtrate should be added (2) a sufficient quantity of a saturated alcoholic solution of fuchsin to give the fluid a dark, almost opalescent appearance (the exact amount of the stain is not important).[1] The prepared cover-glass is floated—specimen side down—on this coloring fluid, and gently boiled from three to five minutes.

The entire specimen is thus completely stained, tubercle bacilli, tissue elements, and other bacteria which may be present, all in the same way. The next step is to remove the color with acid from all the structures which may be intermingled with the tubercle bacilli; the latter, owing to the tenacity with which they retain the color, being but slightly affected. This is done by dipping the cover-glass into an aqueous solution of twenty-five-per-cent nitric acid, and shaking it about for a few seconds. The acid may be even a little more dilute than this. Under the influence of the acid, the specimen on the cover-glass loses its red color and becomes gray or colorless. It is then thoroughly rinsed in three or four successive portions of alcohol, and finally in water. By this manipulation the red color may be to a slight extent restored.

Care should be taken not to expose the specimen too long to the action of the acid, because then the bacilli may be also partially or completely decolorized. A little experience will enable the experimenter to judge of the proper time for the action of the acid.

The specimens may be studied in water with the use of a high-power lens—preferably an oil immersion—and the Abbé condenser, or they may be dried in the air and mounted in balsam.

Inasmuch as not infrequently some other bacteria besides the tubercle bacilli retain a slight red color, it is well, after the specimen is rinsed in water, to float the cover-glass for a few minutes in a dilute aqueous solution of methylin blue, which will replace the red color in all of the bacteria except the tubercle bacilli, thus forming a marked color contrast between them.

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[1] This stain should be freshly prepared, as it does not usually keep well for more than a few days.

Various other aniline dyes may be used instead of the fuchsin, and there are various minor modifications of the process which are often employed; but, on the whole, for routine sputum examinations we recommend the method here given.

In Sections.—Thin sections of tubercular tissue which have been hardened in alcohol are stained in the same way, except that instead of hastening the process by heating, which is apt to shrivel the sections, they must lie in the dye for from twelve to twenty-four hours. After decolorization by acid and dehydration by alcohol, the sections are cleared in oil of cloves and mounted in balsam. In specimens prepared in this way, the bacilli stand out as sharply defined slender rods or filaments on the uncolored or but slightly tinted background (Fig. 129).

Other dilute acids besides nitric acid may be used for decolorization, such as hydrochloric. This is sometimes desirable in examining delicate tissues, since the nitric acid often causes shrinkage and distortion in the specimen. A one-per-cent alcoholic solution of hydrochloric acid is suitable for this purpose.

For purposes of simple recognition of the bacilli in specimens, it seems to the writer usually better to have no color in the preparation other than that which the tubercle bacilli possess. But it is often convenient to demonstrate the nuclei of the cells at the same time, and this may be accomplished by staining afterwards with some color which will contrast with that of the bacilli, such as Bismarck brown or methyl blue.

Cultures.—The tubercle bacillus grows readily in artificial cultures at the temperature of the body, on blood-serum, also on agar to which ten per cent of glycerin has been added, and on boiled potatoes, provided that they be sealed in tubes so that the surfaces may remain moist. It forms on the surfaces of the culture dry, scaly, usually not very voluminous grayish masses.[1]

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[1] *Bibliography.*—Aside from the standard works on general pathology, the reader may consult, for an account of the earlier literature and investigations on tuberculosis up to 1869, the monograph of *Waldenburg*, "Die Tuberculose," Berlin, 1869. The announcement of the discovery of the Bacillus tuberculosis by Koch was made in the Berliner Klin. Wochenschrift, 1882, No. 15. A most elaborate and valuable article on the same subject by *Koch* is contained in the "Mittheilungen aus dem Kaiserlichen Gesundheitsamte," vol. ii.

The very voluminous literature on the subject of the tubercle bacillus which has accumulated since 1882 is for the most part scattered through the German, English, and French journals. It may be best obtained by consulting files of the Index Medicus of dates since April, 1882.

LUPUS.

This form of inflammation most frequently occurs in the skin of the face, but also in the mucous membrane of the mouth, pharynx, conjunctiva, vulva, and vagina. The lesion consists of small, multiple nodules of new-formed tissue, somewhat resembling granulation tissue, in the cutis or mucosa and submucosa. By the formation of new nodules and a more diffuse cellular infiltration of the tissue between them, the lesion tends to spread, and by the confluence of the infiltrated portions a dense and more or less extensive area of nodular infiltration may be

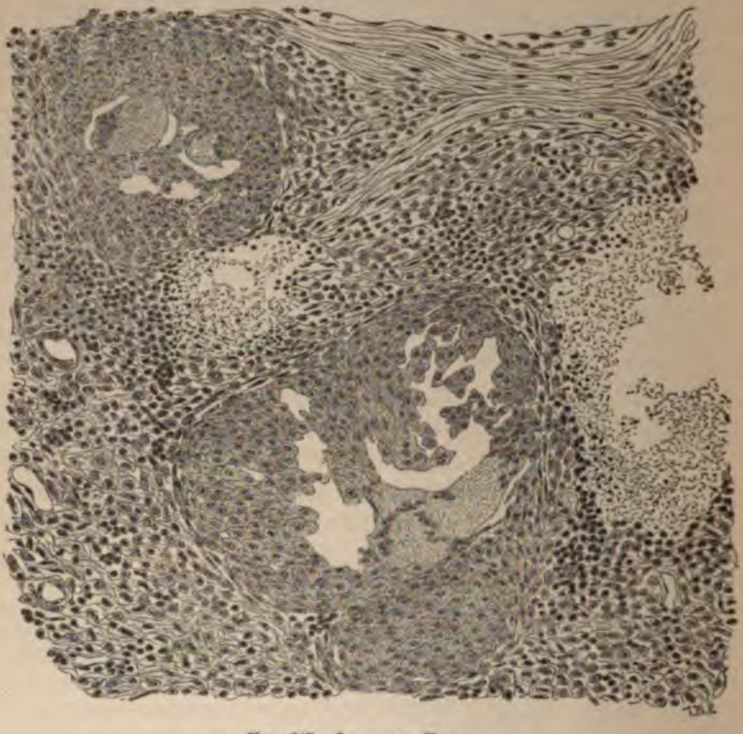

FIG. 217.—LUPUS OF FACE.

formed. There may be an excessive production and exfoliation of epidermis over the infiltrated area, or an ulceration of the new tissue.

Microscopical examination shows the lesion to consist in the formation of tissue composed of small spheroidal cells, intermingled with variable numbers of larger, so-called epithelioid cells and cell masses, and, in many cases, with giant cells (Fig. 217). In some cases, a well-marked reticulum is present between the new cells, and these are often grouped in masses around the blood-vessels. In some cases, there is, without previous ulceration, a formation of new connective tissue in the diseased area, and a well-marked cicatrization; in other cases, the cells and intercellular substance undergo a disintegration which leads to ulceration.

The morphological characters of the lesion long ago led to the conjecture that lupus was in reality a form of tubercular inflammation. This view has now become established by the numerous observations which show the very constant presence of the tubercle bacillus at the seat of inflammation. It is not unlikely, however, that in the clinical group of diseases called lupus there may be lesions which are not caused by the tubercle bacillus. More exact bacterial studies must be made before this point can be fully decided.

ERYSIPELAS.

Erysipelas is a diffuse inflammation of the skin and subcutaneous tissue which tends to spread, and which especially involves the lymph spaces and the lymph vessels. It is characterized locally by swelling of the tissue and a bright red color of the integument. It is regularly accompanied by constitutional disturbances, the most marked of which is fever. The morphological changes at the seat of lesion, as we see them after death, vary considerably in different cases and in different stages of the disease. The redness of the disease usually disappears after death. But the tissues may be swollen by the accumulation of serous fluid. This fluid may be nearly transparent, or turbid from admixture with pus cells. Pus cells may infiltrate the tissues either sparsely or in dense masses. Sometimes vesicles are found on the surface, or scabs; sometimes more or less of the affected region becomes filled with abscesses or gangrenous. In some cases we find, aside from the local lesions, petechiæ in the serous membranes, swelling of the spleen, and parenchymatous degeneration of the kidneys and liver.

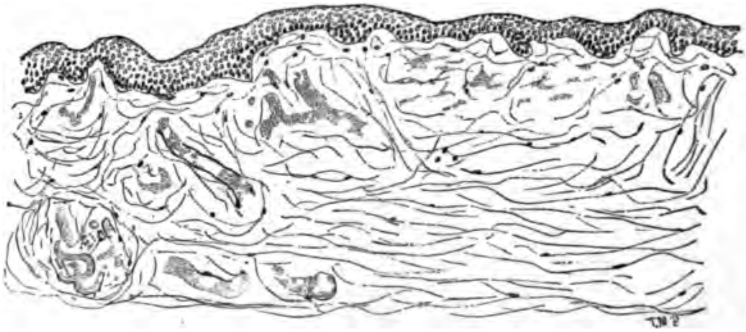

FIG. 218.—MICROCOCCI IN BLOOD AND LYMPH VESSELS OF THE SKIN IN ERYSIPELAS.

The researches of Fehleisen and others have shown that erysipelas is caused by the presence and action in the tissues of a chain coccus called *Streptococcus erysipelatos*. These bacteria are usually most abundant in the lymph vessels and lymph spaces along the advancing borders of the inflammatory area, but they may be contained in the blood-vessels (see Fig. 218).

The Streptococcus erysipelatos is a moderately large coccus, varying, however, considerably in size, and may occur singly, in pairs, or in longer and shorter chains (see Fig. 219). It is readily cultivated artificially, forming in gelatin tubes a series of small whitish colonies along the puncture line, and not fluidifying the gelatin. Its growth in general is slow. It forms a scarcely visible growth. At 35° C. on potatoes, and in beef-tea at the same temperature, it forms within twenty-four to forty-eight hours an abundant flocculent deposit which may cling to the sides of the tube.

Inoculation of rabbits with the pure culture may induce a fairly typical erysipelatous inflammation, but, as these animals are not especially susceptible to its action, the results of inoculations are not constant.

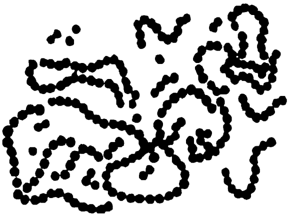

FIG. 219.—STREPTOCOCCUS ERYSIPELATOS. From a beef-tea culture.

In its morphological and histological characters it appears to be identical with the Streptococcus pyogenes and Streptococcus diphtheriæ. There appears to be much reason for believing that many forms of phlegmonous inflammation, and many forms at least of diphtheria and erysipelas, are different phases of the inflammatory process due to the same organism; the difference in the reaction of the tissues which constitute the clinical differences characteristic of the different diseases being due, perhaps to differences in the tissues involved, perhaps to variations in the characters and virulence of the germ, and perhaps to causes which at present we know nothing about.

Further researches are required to fully explain the exact relationship of these at least closely allied forms of inflammation to one another and to the bacteria which cause them.

PYÆMIA.

It has long been known that a certain number of persons who have received wounds by accident or by the hands of the surgeon may suffer from constitutional symptoms and develop lesions. To designate the condition of these patients, the terms pyæmia, septicæmia, septo-pyæmia, ichorrhæmia, inflammatory fever, surgical fever, traumatic fever, suppurative fever, and purulent infection have been used. Attempts have been made to distinguish several different forms of disease and to call one pyæmia, another septicæmia, and so on, but these attempts have not proved satisfactory, and for the present it is better to include them all under the general name of pyæmia.

As regards the nature of pyæmia three theories have been held :

I. That pus is absorbed, circulates in the blood, and acts as a poison.

II. That a chemical poison is evolved from pus and the other matters which are found in wounds, and that the system is poisoned by this.

III. That bacteria are introduced into and developed in the wound, find their way into the blood and tissues, and there multiply (see page 92).

The following are the ordinary forms of the disease commonly called pyæmia, with the lesions :

I. There are cases of wounds and injuries which are characterized by the presence of a febrile movement without any other symptoms. In such cases, no lesions are found except extravasated blood and portions of necrotic tissue, neither of which are in the condition of putrefaction.

II. There are cases in which, in some part of the body, a portion of tissue is not only dead but putrefying. While the process of putrefaction is going on, the patient suffers from rigors, a febrile movement, great prostration, and may die. If, however, the dead tissue can be removed or the putrefactive process arrested, all the symptoms rapidly disappear.

III. A very small wound may apparently introduce at once into the body a specific poison, as in dissecting wounds. There are swelling and redness about the wound, and inflammation of the lymphatic vessels and glands in its neighborhood. Later all the neighboring tissues become involved in an unhealthy inflammation; there may be infarctions in the viscera, a large soft spleen, and parenchymatous degeneration of the liver and kidneys.

IV. There is a large number of cases which it is difficult to classify. They are the ordinary hospital cases of compound fracture and surgical wounds. After the death of these patients, there is a considerable variety in the post-mortem appearances.

1. There are cases in which there are no recognizable lesions.

2. There are cases characterized by early post-mortem decomposition; post-mortem staining of the tissues; congestion of the lungs, stomach, intestines, and kidneys; extravasations of blood in the serous membranes; swelling of the solitary and agminated glands in the small intestine; swelling of the spleen and parenchymatous degeneration of the liver and kidneys.

3. In some cases there are localized inflammations. The joints, the connective tissue around the joints, the pleura, the pericardium, the peritoneum, the pia mater, and the connective tissue in different parts of the body may be inflamed. These local inflammations are of a purulent character, except in the serous membranes, where the principal inflammatory product may be fibrin.

4. There are cases in which the veins in the neighborhood of the wound contain softened, puriform thrombi; there are no infarctions in the viscera, but there may be inflammation of the joints and serous membranes.

5. In other cases the veins contain thrombi; there are infarctions and abscesses in the viscera; local inflammations of the joints and serous membranes may be present or absent. The thrombi are formed regularly in the veins near the wound, but they may be situated in veins at a distance, and sometimes, although infarctions and abscesses are present, no thrombus can be discovered. The veins may be distended by the thrombi or only contain small coagula. The different kinds of thrombi, and the varieties of emboli and infarctions which they produce, are described in the article on Thrombosis, page 55.

V. *Prolonged Suppuration.*—There is first a wound, or a bruise, or an idiopathic suppurative inflammation. This original focus of inflammation continues to suppurate for a long time, then successive abscesses are formed in different parts of the body, the patients lose flesh and strength, and die in a condition of extreme emaciation. After death, abscesses are found in different places, but not in the viscera. There are no thrombi nor infarctions. There may be bronchitis or broncho-pneumonia. The liver, spleen, and kidneys are often the seat of waxy degeneration.

VI. *Spontaneous Pyæmia.*—Under this name we include a group of cases which resemble ordinary pyæmia in their symptoms and lesions, but are of obscure etiology. There is no wound, fracture, or abscess to account for them, but they present the same symptoms and the same lesions as the ordinary cases of pyæmia.

Bacteria in Pyæmia.—In a very large proportion of cases of pyæmia, micrococci, scattered and in colonies, are found in various parts of the body (Fig. 220). The species which have been most frequently identified are the Streptococcus pyogenes and the Staphylococcus pyogenes. These bacteria, when present, apparently stand in a causative relation to the disease. But until we have a more precise conception of what we mean by the name pyæmia, it is difficult to speak with much definiteness of its causative factors.

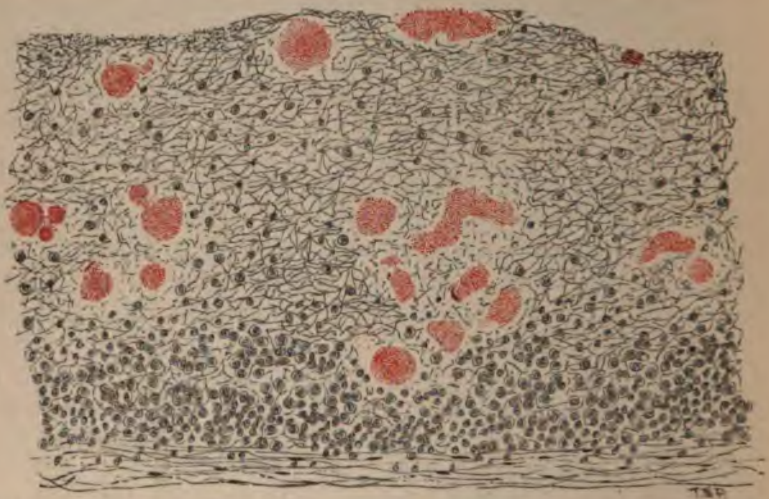

FIG. 220.—MICROCOCCI IN ZOOGLŒA COLONIES IN THE FIBRINOUS EXUDATION OF PYÆMIC PLEURISY.

If we regard pyæmia as an acute infectious disease, caused by the entrance into the body of certain bacteria, then it is probable that the bacteria most frequently concerned are those species above mentioned, which are also the most common causative factors in suppurative inflammation.

ANTHRAX.

Synonyms.—Splenic fever; Malignant pustule; Charbon, Carbuncle.

This disease, which is much more common in the lower animals, especially the herbivora, than in man, is of infrequent occurrence in the United States. It is induced in man by the accidental inoculation with the Bacillus anthracis, which causes the disease in the lower animals. This may occur through the agency of flies which have been feeding on animals infected with this disease, by handling their carcasses or hides, or in other ways. There may be in man local lesions in the skin, in the form of papules, pustules, or a phlegmonous inflammation, with œdema and lymphangitis. The disease may remain local, or general infection may occur. In some cases, there is apparently no external lesion.

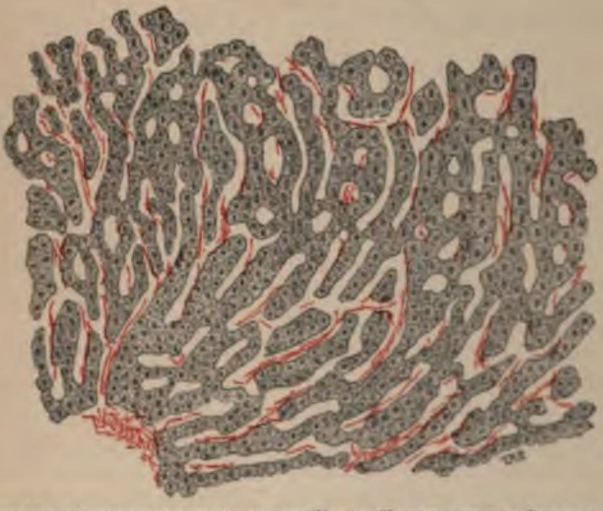

Fig. 221.—Bacillus anthracis growing in the Blood Vessels of the Liver of a Mouse inocculated with a Pure Culture of the Bacillus.

When general infection occurs, the post-mortem appearances vary.

Decomposition, as is usual in acute infections, generally sets in early. The blood is frequently not much coagulated and dark in color. Hæmorrhages and ecchymoses are frequently found in the serous and mucous membranes, and in various other parts of the body.

The lungs may show small hæmorrhages and œdema, and the bronchi may be deeply congested. The pleural cavities may contain serum. The intestines may exhibit the lesions of the so-called *intestinal mycosis* (see page 338). The bronchial and other lymphatic glands may be swollen. The spleen may be swollen, very dark in color, and soft, sometimes almost diffluent.

The bacillus which causes the disease may be found, usually in large numbers, in the spleen and in the capillary blood-vessels, especially in the liver, lungs, kidneys, and intestine (see Fig. 221).

The Bacillus anthracis is from 5 to 20 μ long, and about 1 μ broad, and is often slightly curved (see Fig. 222). The ends are not rounded, and they often hang together end to end, forming thread-like structures. They develop spores outside of the body which are very invulnerable to the action of the ordinary germicidal agents and to heat. They are immobile.

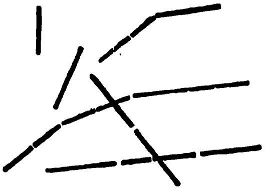

FIG. 222.—BACILLUS ANTHRACIS.

They are readily cultivated outside of the body, and, when thus purified, their inoculation into various species of animals produces the disease, and in the blood of the diseased animals multitudes of the bacilli are found, showing their proliferation in the blood-vessels and elsewhere. They are of especial interest and importance, because we know more of their life history than of almost any other of the bacteria, and because it was this bacterium which was first absolutely demonstrated to be the cause, and the only cause, of a well-defined disease in man.[1]

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[1] For the literature of the relations of the Bacillus anthracis to this disease, etc., see *Koch*, "Mitth. a. d. Kaiserlichen Gesundheitsamte," Bd. 1, p. 49 et seq.

LEPROSY.

Lepra (Leprosy).—This form of inflammation is characterized by the development of nodular and sometimes diffuse masses of tissue, consisting of larger and smaller cells of various shapes—spheroidal, fusiform, and branched—the whole somewhat resembling granulation tissue. The new tissue is most frequently formed in the most exposed parts of the skin, as the face, hands, and feet, but it may occur in the skin of any part of the body. It is formed more rarely in the subcutaneous connective tissue, in intrafascicular connective tissue of nerves, in the viscera, and in the mucous membranes. The mucous membranes most frequently affected are those of the eye, nose, mouth, and larynx. The nodules may be very small or as large as a walnut, and may be single or joined together in groups or masses. The tissue of the part in which the new formation occurs may be atrophied and replaced, or may remain intermingled with the leprous tissue, or it may be hypertrophied. The nodules may persist for a long time without undergoing any apparent change, or they may soften and break down, forming ulcers; but ulceration, except in the mucous membranes, is said usually to occur as the result of injury or unusual exposure. The leprous tissue may change without ulceration into cicatricial tissue, or cicatrization may follow ulceration.

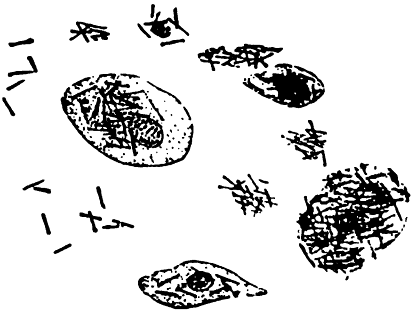

FIG. 223.—THE BACILLI OF LEPROSY.
Stained with fuchsin. From a nodule in the skin.

Various secondary lesions and disturbances of nerve function are associated with the formation of leprous tissue in the nerve and central nervous system, but these we cannot consider here.

In all the primary lesions of leprosy, bacilli are said to be present, mostly in the cells, and particularly in the larger spheroidal forms, but sometimes free in the intercellular substance. The bacilli have been found in the skin, mucous membrane of the mouth and larynx, in peripheral nerves, in the cornea, in cartilage, in the testicles, and in lymph nodes. Sometimes the cells contain but few bacilli, but they are frequently crowded with them. The bacilli are from 4 to 6 μ long and very slender, being usually less than 1 μ in thickness. They are sometimes pointed at the ends, and sometimes present spheroidal swellings, which seem to indicate the formation of spores (Fig. 223). They are capable of spontaneous movement. In their comportment towards staining agents, as well as in general morphological characters, they considerably resemble the Bacillus tuberculosis. They may be stained with fuchsin or gentian violet by the ordinary method, or by the method employed for staining the tubercle bacillus.

According to Neisser, the lepra bacillus may be artificially cultivated on blood-serum and on boiled eggs.

The reasonable conjecture that the Bacillus lepræ causes the lesions of leprosy depends as yet largely upon the constancy of their occurrence in the disease, and upon their relations to the cells of the new growth.

GLANDERS—FARCY.

These names are given to two varieties of the same disease. The disease originates in the horse and occurs in men by contagion. According to some authors, it may be idiopathic in the human subject.

In the horse we find four varieties of the disease: chronic and acute glanders, chronic and acute farcy.

1. *Chronic Glanders.*—The disease begins in the mucous membrane of the nose. Small, whitish nodules, composed of small round cells, are formed in the mucous membrane. These nodules soften and ulcerate. The same nodules may be found in the larynx, trachea, and bronchi. The ulcerations may remain superficial, or they may extend and attack the subjacent cartilage and bone. Nodules of the same kind may be found in the lungs.

2. *Acute Glanders.*—There is the same formation of nodules, which soften and ulcerate; but they are accompanied with an intense inflammation of the nasal mucous membrane, and the disease runs a rapid course.

3. *Chronic Farcy.*—The lymphatic glands and vessels become enlarged, and nodules are formed in the skin, lungs, and other viscera. The glands become cheesy or soften and suppurate. The cutaneous nodules soften and suppurate.

4. *Acute Farcy.*—There are the same lesions of the lymphatics, and nodules and abscesses are found in the skin. There are also nodules in, and inflammation of, the nasal mucous membrane, and the disease runs an acute course.

In man, the disease occurs in an acute and a chronic form, but does not exactly resemble any of the varieties of the disease in the horse.

The acute disease runs a rapid and malignant course. The skin may be covered with a pustular eruption. Furuncles, carbuncles, and abscesses are found beneath the skin and in the muscles. Nodules are found in the nasal mucous membrane, the lungs, kidneys, testes, spleen, and liver. The joints may be inflamed, and there may be osteomyelitis.

The disease may begin at a single point, so that it may be mistaken for a carbuncle or a gangrenous erysipelas. Graefe gives a ca[illegible]h began as an acute exophthalmos, and the nature of the disease was not ascertained until after death. In this case there were nodules in the choroid coat of the eye.

The chronic form of the disease is still more difficult of diagnosis. The nodules grow very slowly, are hard, and may occur in groups or like a string of beads. The nodules may soften and form chronic ulcers.

A slender bacillus, called *Bacillus mallei*, has been proven to be the cause of glanders both in man and animals. They occur either scattered or in masses in the areas of fresh inflammation, and have been found in the blood of affected individuals. They have been repeatedly artificially cultivated, and successful inoculations practised with the pure cultures upon animals. The bacilli grow at the temperature of the body on blood-serum and on potato pulp.[1]

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[1] Consult *Loeffler*, "Arbeiten a. d. Kais. Gesundheitsamte," Bd. 1, 1886.

HYDROPHOBIA.

The lesions which have been found in this disease are not constant nor are they characteristic. Though well marked in some cases, they are but very slightly developed in others.

The lesions, when present, are apt to be most marked in the medulla oblongata and pons, but they may be present in the cord. They consist of small hæmorrhages, and accumulation of leucocytes about the blood-vessels in the perivascular lymph spaces and of thrombi in the smaller blood-vessels.

While there is much reason for believing that hydrophobia is due to the introduction into the body of some special form of micro-organism, and while the recent researches of Pasteur and others have brought to light many interesting and important facts regarding the general nature and distribution in the body of the infectious agent, nothing is yet definitely known about the particular organism which induces the disease.

THE MALARIAL FEVERS.

The characteristic lesions of malarial poisoning are certain changes in the blood, the spleen, and the liver.

In the more intense and acute form of malarial poisoning, the blood contains numerous particles of black or brown pigment, which are either free or imbedded in cells resembling the white blood-cells and the endothelium of the blood-vessels (see Fig. 164). After death, this pigment is found in the blood-vessels throughout the body, but is most abundant in the blood-vessels of the liver and spleen. These organs are then usually of large size and of a peculiar brown or black color.

In some of these severe cases, there are also extravasations of blood from the mucous membranes, and in their substance. There may also be general jaundice.

In the milder and more protracted cases of malarial poisoning, the composition of the blood is altered, and the patients may become profoundly anæmic. The spleen may become the seat of chronic interstitial inflammation with pigmentation (see Fig. 174). The liver may exhibit the changes of chronic interstitial hepatitis.

The attempts to establish a causative relationship between the various forms of bacteria which from time to time have been found in the bodies of persons who are the victims of malarial poisoning, and the symptoms and lesions of the disease, have all been unsuccessful.

On the other hand, a large number of careful studies by various observers have led to a strong presumption that the disease is due, not to a vegetable, but to an animal organism which is very constantly found in the blood of affected persons.

In brief, the facts upon which this presumption rests are as follows: The blood of those suffering from malarial poisoning may contain one or more of the structures which are shown in Fig. 224.

1. Inside of the red blood-cells may be found colorless bodies, sometimes occupying a small part, sometimes nearly filling the cell. These bodies may or may not contain pigment granules. They may exhibit amœboid movements (*a* and *b*). They are called the ***amœboid bodies.***

2. Colorless discoidal bodies, usually a little larger than the red blood-cells, which contain pigment particles, sometimes scattered irregularly, sometimes grouped towards the centre. These are believed by some observers to be later developmental stages of the amœboid bodies, which have increased in size at the expense of the red blood-cell. A grouping of the pigment granules indicating segmentation is sometimes seen in these bodies. These are called the *incysted bodies* (*c*).

3. Bodies, about the size of a red blood-cell, which are composed of a congeries of irregularly rounded structures grouped about a central mass of pigment. These are called *segmenting bodies* or *rosettes* (*d*).

4. Smaller isolated or clustered structures which are apparently the result of the breaking apart of the segmenting bodies as seen at *e*. Often called *spores*.

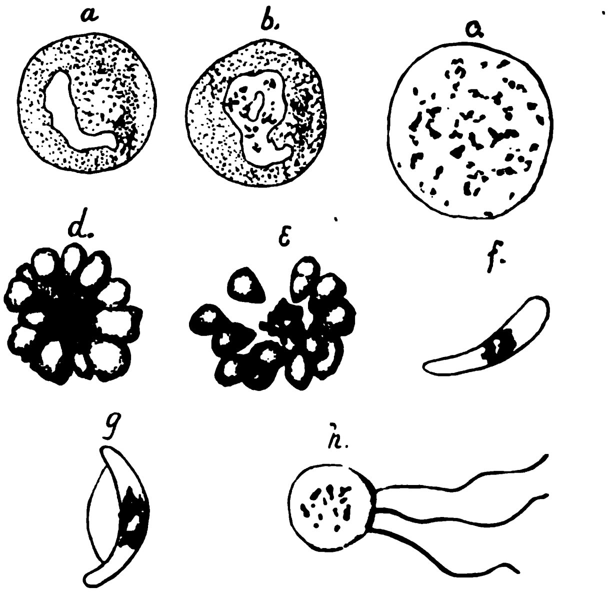

FIG. 224.—PLASMODIUM MALARIÆ IN THE BLOOD.

a, unpigmented amœboid body in a red blood cell; *b*, pigmented amœboid body; *c*, colorless discoidal body with pigment; *d*, segmenting body; *e*, fragments of segmenting body; *f* and *g*, crescentic bodies; *h*, flagellate body. *a*, *b*, *d*, *e*, *f*, *g*, are drawn from specimens of malarial blood prepared by Dr. Walter James; *c* and *h* are drawn after sketches by Dr. James.

5. *Crescentic* bodies containing a central mass of pigment (*f* and *g*).

6. Bodies, smaller than a red blood-cell, which are actively mobile and are furnished with one or more flagellæ at one side—*h*, *flagellate form*. These are the main forms which have been described.

The amœboid forms are apt to occur in the acute stages of the disease, the crescentic forms in the chronic stages. The segmenting bodies are apt to be present immediately before or during the chill; the pigmented amœboid bodies, according to James, are present at all times, but are most numerous during the paroxysm. The amœboid forms disappear shortly after the administration of quinine, while the crescentic bodies often persist for a considerable time under the same conditions.

Whether these various bodies are developmental forms of the same organism or not has not yet been fully established, as artificial cultivations have not been made.

The organism, which apparently belongs among the protozoa, may be most appropriately called the *hæmatozoon of malaria*. It is, however, often called the Plasmodium malariæ.

Whatever its etiological significance or its life history, its discovery in the blood, even with our present knowledge, since it is unknown except in malarial disease, is of great diagnostic value in doubtful cases.

Method of Examination.—The fresh blood taken from a finger prick may be examined in thin layers with one-twelfth oil immersion. Or the blood may be spread in a very thin layer on a cover-glass, dried, passed through a flame, and then stained as in the ordinary mode of bacterial examination with fuchsin or methylin blue (see page 96). The stained specimens may be mounted and preserved in balsam.[1]

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[1] For a more detailed account of the hæmatozoon of malaria and literature, consult *James*, "Micro-organisms of Malaria." Proceedings New York Pathological Society, January 25th, 1888.

DISEASES CHARACTERIZED BY ALTERATIONS IN THE COMPOSITION OF THE BLOOD.

There is a group of diseases in which the essential lesion seems to be an alteration in the composition of the blood, although in some members of the group other lesions are also present. This group embraces Chlorosis, Pernicious Anæmia, Addison's Disease, Leukæmia, and Pseudo-Leukæmia.

CHLOROSIS.

In many cases of chlorosis the only lesion is the change in the composition of the blood.

The volume of the blood may be diminished, the red blood-globules contain less hæmoglobin, the number of red blood-cells is diminished; there are present red blood-cells, some of which are larger and some smaller than the normal cells.

In a few cases there is also present a congenital smallness of the aorta.

PERNICIOUS ANÆMIA.

In this disease the same changes in the blood exist as in chlorosis, and there may also be a moderate increase in the number of the white blood-cells.

There is often fatty degeneration of the walls of the heart and of the arteries.

There may be thrombosis of the pulmonary artery.

The same changes in the marrow of the bones are found as in leukæmia.

There may be ecchymoses in different parts of the body.

ADDISON'S DISEASE.

This name is applied to a disease characterized by a peculiar pigmentation of the skin, certain changes in the suprarenal capsules, and a diminution in the number of red blood-cells. The patients become very anæmic, but are not emaciated. They suffer from cerebral symptoms, great prostration, syncope, and derangements of the functions of the stomach and intestines.

The pigmentation of the skin is the symptom which has especially attracted attention. The change in color usually begins and becomes most marked in those parts of the skin which are not covered by the clothing, or are naturally darker colored. The rest of the skin afterwards changes color, but not uniformly, white patches being left. The color is at first a light yellow or brown; this becomes darker until it is of a dark greenish, grayish, or blackish brown. The mucous membrane of the tongue, lips, and gums may be pigmented in the same way.

Under the name of Addison's disease different observers have described cases in which the symptoms and bronzed skin existed without disease of the suprarenal capsules; cases in which the bronzed skin was the only lesion, and cases in which the suprarenal capsules were diseased without symptoms or bronzed skin.

We hardly know as yet what are really the characteristic lesions of the disease.

The Skin.—The discoloration of the skin is due to deposit of yellowish-brown pigment in the deeper layers of the epidermis, especially in the layer covering the papillæ, and less constantly in the connective tissue of the cutis.

The Brain.—Pigmentation of the gray matter, acute meningitis, chronic meningitis, and distention of the ventricles with serum have been observed.

The Heart.—The muscular fibres may be the seat of fatty degeneration.

The Sympathetic Nerves may show a variety of changes apparently due to chronic inflammation, especially the nerves which are in contact with the suprarenal capsules.

The Suprarenal Capsules.—The most common lesion of these bodies is a tubercular inflammation resembling that which occurs in the lymphatic glands.

The suprarenal capsules are large, hard, and nodular; less frequently of normal size or atrophied. On section, they are found to contain cheesy masses, surrounded by zones of gray, semi-translucent tissue. Later the cheesy masses may become calcified or they may soften and break down.

The grayish zones are composed of tubercle tissue, granulation tissue, and connective tissue.

Other cases have been described in which the suprarenal capsules were the seat of carcinoma, or of fatty or waxy degeneration. The suprarenal capsules in some cases appear normal.

The Blood exhibits the same changes as exist in pernicious anæmia.

LEUKÆMIA (LEUCOCYTHÆMIA).

This disease is characterized by a persistent and progressive increase in the number of white blood-cells in the blood, accompanied by alterations of varying amount in the spleen, lymph glands, and bone marrow. Sometimes one, sometimes another of these organs is especially involved. Not infrequently other internal organs, lungs, liver, kidneys, etc., are more or less densely infiltrated with leucocytes, either diffusely or in nodular form. The lymphatic tissue of the gastro-intestinal canal may be in a condition of hyperplasia.

Ecchymoses in the serous and mucous membranes, or severe hæmorrhages on slight provocation, and fatty degeneration of the heart and kidneys, frequently complicate leukæmia. Aside from various other foreign chemical substances which may exist in the blood in leukæmia, there are very frequently found in the blood, marrow, spleen, liver, etc., after death, elongated octahedral crystals, called *Charcot's crystals*, which are believed to be formed by a combination of phosphoric acid with some organic base.[1]

For a detailed description of the lesions of the different parts of the body in leukæmia, see chapters on Blood, Spleen, Lymph Glands, Bones, etc. It does not fall within the scope of this work to consider the various theories as to the probable causes of this disease, which is very obscure and but little understood.[2]

PSEUDO-LEUKÆMIA (HODGKIN'S DISEASE).

In *pseudo-leukæmia*, which is very closely related to leukæmia, the anatomical changes in the organs are apparently identical with those of leukæmia, but there is no increase in the number of leucocytes in the blood. Of the internal organs the lymph glands are most apt to be specially affected in pseudo-leukæmia.

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[1] For literature of Charcot's crystals, consult *Zenker*, Arch. für klin. Medicin, Bd. 18, p. 125, 1876.

[2] For literature of leukæmia and pseudo-leukæmia, consult *Birch-Hirschfeld*, "Lehrbuch der path. Anatomie," 2d ed., Bd. 2, p. 146. For general consideration of the relation of certain diseases of the blood to the blood-forming organs—spleen, lymph glands, etc.—see *Cohnheim's* "Vorlesungen über allgemeine Pathologie," vol. i.

SCORBUTUS—PURPURA—HÆMATOPHILIA.

SCORBUTUS (SCURVY).

This disease appears to result from imperfect nutrition under conditions which cannot be considered here, and whose immediate cause we do not understand. The lesions are variable, the most prominent being extravasation of blood in the skin, subcutaneous tissue, and muscles; swelling and ulceration of the gums. Small and sometimes extensive hæmorrhages are apt to occur in the mucous membranes and on serous surfaces. Small ulcers may form in the mucous membranes. Fatty degeneration of the heart, liver, and kidneys is not uncommon. The spleen may be large and soft. No constant characteristic changes have been discovered, either in the blood-vessels or the blood, which would satisfactorily account for the extravasations and other lesions.

The body is apt to decompose early. The skin may be mottled with small and large purple, blue, brown, or blackish spots produced by degenerative changes in the extravasated blood in the cutis. Sometimes ulcers are produced by the perforation of effused blood on to the surface.

The joints may be inflamed, may contain serum or blood. Rarely the hæmorrhages are followed by destruction of the cartilages and ends of the bones.

Very rarely there is hæmorrhage between the periosteum and bone, and in the bone itself, producing softening and destruction of the bone, and separation of the epiphyses. The sternal ends of the ribs are the most frequent seat of this change.

PURPURA HÆMORRHAGICA (MORBUS MACULOSUS).

This disease is characterized by the occurrence of ecchymoses in the skin, mucous and serous membranes. Hæmorrhages, particularly from the mucous membranes, may be very severe and even fatal. The cause of the disease is unknown.

HÆMATOPHILIA (HÆMORRHAGIC DIATHESIS).

This disease consists in a liability to hæmorrhage on the slightest provocation, and is dependent upon some constitutional peculiarity which is unknown to us. It is frequently hereditary. An unusual thinness of the intima of the arteries has been noticed in some cases, and other changes have been described, but there are no constant lesions associated with the hæmorrhages, as yet discovered, which would satisfactorily explain their occurrence. The hæmorrhages may be traumatic in origin, or they may occur spontaneously from the mucous membranes.

P. K. H.

GOUT.

The characteristic lesion of gout is the deposit of urate of soda in the articular cartilages, the ligaments of the joints, the ears, and the eyelids.

The most frequent situation is the metatarso-phalangeal joint of the great toe. The cartilage may be infiltrated or incrusted with the deposit.

A very important feature of gout is that patients with the gouty diathesis are especially liable to derangements of digestion and to certain chronic inflammations, such as chronic inflammation of the arteries, chronic bronchitis, and chronic nephritis.

DIABETES.

It would be expected that so common and well marked a disease as saccharin diabetes should be characterized by definite lesions, but this is not the case. The changes which we find after death are accidental, or the results of the disease. No characteristic lesions have yet been discovered.

The Brain may appear to be entirely normal; it may be congested; there may be an increase of serum; the convolutions may be shrunken; there may be meningitis; there may be dilatation of the blood-vessels, small extravasations of blood around the vessels, enlargement of the perivascular spaces, and alterations in the perivascular sheaths, and nervous matter bounding the cavities; there may be tumors at the base of the brain.

The Spinal Cord may present dilatation of the blood-vessels; dilatation of the central canal; changes in the gray matter of the anterior cornua.

The Lungs.—There may be pleurisy, bronchitis, broncho-pneumonia, lobar pneumonia, gangrene of the lung, chronic pulmonary phthisis.

The Heart is often small; there may be chronic endocarditis.

The Stomach and Intestines.—The stomach may be dilated, its walls may be thickened, there may be hæmorrhagic erosions of the mucous membrane. In the intestines there may be tubercular ulcers or enteritis.

The Liver and the Spleen show no marked changes.

The Pancreas may be atrophied; it may contain abscesses.

The Kidneys may be enlarged; they may be the seat of parenchymatous or diffuse nephritis; there may be glycogenic degeneration of the epithelium of Henle's loops.

The Blood.—In a few cases fat has been found in the blood and fat emboli in the vessels of the lungs.

A considerable number of autopsies are recorded in the Transactions of the Pathological Society of London, vol. xxxiv., p. 328, and in Frerich's monograph on "Diabetes."

SUNSTROKE.

During the hot summer months, cases of sunstroke are of frequent occurrence in New York. The persons affected are, for the most part, adult male laborers, usually of intemperate habits.

It is necessary to separate from the cases of sunstroke proper, when the patient is attacked while exposed to the heat of the sun, the cases of exhaustion from heat and fatigue, which may occur as well in the house.

The patients who are seriously affected by sunstroke exhibit, during life, an intense heat of the skin, convulsions, and coma. Death in many cases soon ensues. In other cases, the symptoms are more protracted.

After death, decomposition sets in very early, owing to the state of the weather. In autopsies which I have made within two hours after death, the increased heat of the skin was still maintained.

The Brain and its membranes were in some cases congested, in others not. Sometimes there was an increased amount of serum beneath the pia mater; sometimes there were small and thin extravasations of blood beneath the pia mater, and between the pia and dura mater.

In the other viscera there were no lesions except those due to the condition of coma existing before death. The lungs and kidneys were frequently congested.

In the cases in which cerebral symptoms are protracted for a number of days, the lesions of meningitis have been found after death.

Attention has been called by Dr. H. C. Wood, Jr., to the rigid condition of the wall of the heart after death, but this rigidity is certainly not present in all cases.

DEATH FROM BURNING.

Death may be caused by the inspiration of smoke and flame; by drinking of hot fluids; by the direct contact of flame or hot substances with the external surface of body. It may be due to the direct effect of the agents, to secondary affections of the viscera, or to the exhaustion produced by long-continued inflammation and suppuration.

The entire body may be burned to a coal or completely roasted, or only a larger or smaller area of the skin be burned.

We find the burned skin divested of epidermis and presenting a peculiar red, hard, parchment-like appearance. If the patient has lived some time, this is replaced by a suppurating surface. Or there are small, bladder-like elevations of the epidermis. The base of these blisters is red, and they are surrounded by a red zone, or suppuration may have commenced.

These appearances cannot be produced by heat applied to the skin after death.

The Brain may be congested, œdematous, or softened. More frequently it is normal.

The Larynx and Trachea may be congested and the seat of croupous inflammation. There may be œdema of the glottis.

The Lungs may be congested and œdematous, or hepatized, or the seat of pyæmic infarctions. There may be pleurisy.

Inflammation of the *peritoneum* is not very infrequent. There may be swelling of the *solitary* and *agminated glands* of the small intestine.

The duodenum may be the seat of perforating ulcers and the mucous membrane of the entire gastro-intestinal canal may be congested. The *Liver, Spleen,* and *Kidneys* may be the seat of parenchymatous degeneration or of pyæmic infarctions.

DEATH FROM LIGHTNING.

Persons who are struck by lightning may die instantly; or may continue for several hours comatose or delirious, and then either die or recover; or they may die after some time from the effects of the burns and injuries received.

The post-mortem appearances are very variable. Sometimes there are no marks of external violence or internal lesions. Sometimes the clothes are burnt and torn, while the skin beneath them is unchanged. Usually there are marks of contusion and laceration, or ecchymoses, or lacerated, punctured wounds, or fractures of the bones, or superficial or deep burns. The track of the electric fluid may sometimes be marked by dark-red arborescent streaks on the skin. Fractures are rare.

The internal viscera may be lacerated and disorganized from lightning.

DEATH FROM SUFFOCATION—ASPHYXIA.

By suffocation we understand that condition in which air is prevented from penetrating into the lungs without direct pressure on the larynx or trachea. The interruption of the function of respiration which is thus brought about induces the condition known as *asphyxia*. Many deaths from drowning and strangulation take place in this way.

The methods in which the supply of air may be cut off from the lungs are very various. The mouth and nose may be closed by the hand, by plasters and cloths, by wrapping up the head in cloths, by covering the face with earth, hay, grain, etc. Foreign bodies may be introduced into the mouth, pharynx, and larynx. Blood may pass into the trachea from an aneurism or from a wound. The glottis may be closed by inflammatory swelling. Matters which are vomited may lodge in the larynx.

On the other hand, injury or disease of the medulla oblongata, or paralysis, or spasm of the muscles of respiration from drugs, tumors pressing upon the air passages, or diseases of the lungs themselves, may induce asphyxia.

EXTERNAL INSPECTION.

The body should be examined for marks of violence, the cavities of the mouth and nose for foreign substances.

The face may be livid and swollen or present a natural appearance. The conjunctiva may be congested and ecchymotic. There may be small ecchymoses on the face, neck, and chest. The mouth often contains frothy blood and mucus. The tongue may be protruded.

INTERNAL EXAMINATION.

The Brain and its membranes may be congested, or anæmic and œdematous, or unchanged.

The Blood throughout the body is unusually dark-colored and fluid.

The Larynx may contain foreign bodies which have produced the suffocation. The mucous membrane of the larynx, trachea, and bronchi is congested and sometimes ecchymotic. These passages contain frothy blood and mucus.

The Lungs are usually congested and œdematous, [illegible]times do not differ from their ordinary appearance. There may be small patches of emphysema near the surface of the lungs. Sometimes, especially in infants, small ecchymoses are found in the costal and pulmonary pleura.

The Heart usually presents its right cavities full of blood, its left cavities empty; but tò this there are frequent exceptions.

The Abdominal Viscera are usually congested.

DEATH FROM STRANGULATION—HANGING.

Strangulation is effected by the weight of the body in hanging, by pressure on the neck with the hands or by some other object, or by constriction of the neck with a cord or ligature of some kind. Death is usually produced by asphyxia, or by asphyx a combined with the effect of the cutting-off of the blood supply to the brain by pressure on the large vessels of the neck. In some cases of hanging, death ensues as a result of fracture or dislocation of the cervical vertebræ.

EXTERNAL INSPECTION.

The face may be livid and swollen, the eyes prominent, the lips swollen, and the tongue protruded. These appearances are, however, often absent. Erection of the penis, ejaculation of semen, and evacuation of fæces and urine are frequently observed.

In most cases, marks are left upon the neck by the objects which have directly produced the strangulation.

In cases of hanging, the mark about the neck varies considerably in position, direction, and general characters, depending upon the kind of ligature employed, the time of suspension, period after death at which the observation is made, etc. The most common mark left by a cord about the neck is a dry, dense, brownish furrow, whose breadth corresponds but in a very general way with the diameter of the cord. In some cases, according to Tidy and others, there may be no mark at all if the hanging is quickly accomplished with a soft ligature and the body cut down immediately after death. There may be abrasions and ecchymoses of the skin at the seat of ligature.

In cases of strangulation by the fingers, the marks on the neck may correspond in a general way to the shape of the fingers.

The application of the same forces immediately after death may produce the same marks as when death is induced by them.

INTERNAL EXAMINATION.

The Brain and its membranes may be congested, or there may be extravasation of blood, or there may be no abnormal appearances.

The Neck.—In some cases there is effusion of blood beneath the ligature, rupture of the cervical muscles, fracture of the os hyoides and cartilages of the larynx. fracture and dislocation of the cervical vertebræ, rupture of the internal vertebral ligaments and of the inner and middle coats of the carotid arteries. Similar changes may be produced in the dead body by the use of great violence. In death from asphyxia, the lesions are similar to those described above. In some cases, for example, where death has occurred from fright or shock, the results of post-mortem examination are entirely negative.

DEATH FROM DROWNING.

In examining the bodies of persons who have been drowned it is necessary to bear in mind a number of questions which may arise: Whether the person came into the water alive or dead? How long a time has elapsed since death? Whether the person committed suicide, or was drowned by accident, or was murdered? These questions are to be solved sometimes certainly, sometimes with probability, sometimes not at all, by the post-mortem examination. Persons dying in the water, to which condition the term drowning is commonly applied, may die from asphyxia, from exhaustion, from fright or syncope, from diseases of the heart, apoplexy, injuries, etc. While in the majority of cases asphyxia is a predominant or important factor in death by drowning, the conditions under which death occurs are so apt to be complex that in the minority of cases only are the lesions of pure asphyxia found after death, while in most cases the bodies present the more or less well-marked lesions of asphyxia together with those indicative of complicating conditions. There are no post-mortem conditions which alone are absolutely characteristic of drowning, and it is only by considering all the facts elicited by the autopsy together that any just conclusion can be arrived at. It should always be borne in mind, moreover, that even the most characteristic of the evidences of drowning are apt to be modified or to disappear as decomposition goes on.

EXTERNAL INSPECTION.

Post-mortem rigidity usually sets in early, sometimes immediately after death. Decomposition goes on, especially in summer, with unusual rapidity in bodies which have been removed from the water. Frequently, but by no means constantly, the peculiar roughening of the skin, known as goose skin (cutis anserina), is found, but this may occur after death from other causes. A light, lathery froth, either white or blood-stained, is frequently seen about the mouth and nostrils within twelve to twenty-four hours after removal of the body from the water, but it may be absent, and may be seen after death from other causes. After the body has lain for several hours in the water (twelve to twenty-four), the thick skin of the palms of the hands and soles of the feet may become macerated and thrown into coarse wrinkles, just as it may after prolonged soaking during life, or in a dead body thrown into the water. The penis and nipples may be retracted and the scrotum shrunken, but this is not constant nor characteristic.

If the person has struggled in the water and clutched at objects within his reach, there may be evidences of this in excoriations of the fingers or in the presence of sand, weeds, etc., under the nails or grasped in the hands.

External marks of injury, bruises, etc., should be sought for, since persons in diving, or on being thrown into the water with homicidal intent, may have died from the violence, and not, strictly speaking, from drowning. It should also be borne in mind in such complex cases that injuries, not in themselves fatal, may, when the body is in the water, prove so on account of the inability of the person to rescue himself or gain time for recovery from the injury, and that then the struggle for breath may be but slight, and the more prominent signs of drowning but little marked.

INTERNAL EXAMINATION.

The Brain.—Congestion of the brain and its membranes is found only in a small proportion of cases.

The Blood, when death occurs from asphyxia, is usually fluid throughout the body and of a dark color, as in asphyxia from other causes.

The Air Passages.—In persons who die from asphyxia, the mucous membrane of the larynx, trachea, and bronchi is usually congested, and the air passages contain a variable quantity of bloody or mucous froth. In persons dying in the water from other causes than asphyxia, these appearances are absent. Foreign substances from the water, such as sand, weeds, etc., or matters regurgitated from the stomach, may find their way into the air passages during the act of drowning or as a postmortem occurrence. Thus in bodies washed about on the bottom, sand or mud may get into the air passages for a certain distance, from the mechanical action of the water.

The Lungs in typical cases are distended, so that they fill the thorax and cover the heart. The increased size is due partly to congestion, partly to the presence of the fluid in which the person was drowned, which is often inspired during the act of drowning, and partly to the distention of the air vesicles with air. While in cases of drowning in which there is a struggle, and water is breathed in, the lungs contain more or less fluid, this may, as a result of decomposition, find its way in greater or less quantity into the pleural cavities by transudation, leaving the lungs comparatively empty. It should be remembered, however, that a considerable quantity of reddish fluid may collect in the pleural cavities under other conditions than drowning, as a post-mortem change, by transudation from the blood-vessels and other adjacent tissues.

The Heart.—In those who die from asphyxia, the right cavities are usually filled with fluid blood, while the left cavities are empty. But where death is due to complex causes this may not be the case.

The Stomach.—The fluid in which the person was drowned, sometimes mixed with sand, weeds, etc., may be swallowed during the act of drowning. Sand may wash for a short distance into the œsophagus after death, in bodies washing about the bottom.

The Abdominal Viscera may be congested in persons who die from asphyxia.

In persons dying from syncope, shock, etc., we may find no lesions. When the death is partly due to asphyxia, and partly to other causes, the conditions will vary in various ways, which need not be described in detail here.

In important cases of doubtful drowning, it is desirable to carefully collect and save some of the fluid from the lungs and stomach for micro-chemical examination, since the identification of these fluids with those in which the person was presumably drowned will often give certainty to an otherwise doubtful case.

For the detailed consideration of the anatomical diagnosis of drowning, the changes which bodies dead from drowning undergo from decomposition, and the factors bearing on the question of suicide, homicide, etc., we refer to works on medical jurisprudence.[1]

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[1] *Tidy*, "Legal Medicine," vol. ii., 342–373. *Guy and Ferrier*, "Forensic Medicine," 274–285.

DEATH FROM POISONING.

In cases of suspected poisoning which may possibly have a medico-legal bearing, the examination should be made with extreme care and thoroughness. The inspection of the body and the examination of *all* the viscera should be thorough and detailed. Every appearance should be noted at the time and nothing left to the memory. It is well to have an assistant record the observations as they are made. The disposition of the parts and organs in jars should also be noted at the same time.

It is important to remember that many poisons destroy life without producing appreciable lesions, and also that many cases of sudden death occur, not due to poisons, and without any discoverable cause.

In bodies which are exhumed for examination, the tissues may be so changed by decomposition that it is impossible to say whether lesions have or have not existed. In such cases, the careful and separate preservation of the viscera and other parts for chemical examination is often all that can be done. For directions for preserving tissues and organs for the chemist in medico-legal cases, see Part I. (p. 35).

SULPHURIC ACID.

The effects of this poison vary with the amount taken and with its strength. Death usually takes place in from two to twenty-four hours after the taking of the concentrated acid. A case of death within an hour is recorded. When the poison is less concentrated, or its effects less intense, the patient may survive for months.

The skin of the face about the mouth may be blackened and charred by the acid. The mouth and pharynx are of a grayish or blackish color, or are covered with a whitish layer, while the deeper tissues are reddened. Sometimes these regions escape the action of the poison.

The *larynx, trachea,* and *lungs* are sometimes acted on, softened and blackened by the accidental passage of the acid into them. This may even take place when the acid does not pass into the œsophagus.

The *œsophagus* seldom escapes. It is grayish or blackish colored, softened, and the mucous membrane comes off in shreds. If life is prolonged, cicatrices and strictures are formed. The *stomach* may contain a blackish, pulpy fluid, due to the action of the acid on mucus, blood, etc. It is coated on its internal surface with a black, sticky layer, beneath which the mucous membrane is reddened. The mucous membrane may be blackened in patches or stripes. The organ may be contracted and the mucous membrane corrugated. Sometimes perforation takes place, and the acid blackens and softens the adjoining viscera. In protracted cases, cicatrices are formed and the organ is contracted. If the poison is dilute, there may be only the lesions of chronic gastritis.

The *blood* is sometimes thickened, syrupy, acid, and may form thrombi in the vessels.

The body may be partially preserved from decomposition, owing to the action of the acid upon the tissues.

Fatty degeneration of the renal epithelium is mentioned by some authors.

The solution of indigo in sulphuric acid, commonly known as sulphate of indigo, produces the same lesions as sulphuric acid, and also stains the tissues with which it comes in contact of a dark-blue color. It is stated that an indigo blue tint is often found in the mucous membranes after poisoning by pure sulphuric acid.[1]

NITRIC ACID.

Death may occur very soon after the taking of the poison, but does not usually occur for several hours, and may not take place for several days or weeks.

The surface of the mucous membrane of the *mouth, pharynx*, and *œsophagus* is covered with yellow eschars wherever the acid has touched it. Beneath and around the eschars the tissues are congested and red. The poison may be introduced into the œsophagus without acting on the mouth. The *stomach* contains a viscous, sanguinolent, yellow or greenish fluid. The mucous membrane is congested, red, swollen and softened, ecchymotic. It is rarely perforated. The *duodenum* may be inflamed, and the inflammation extend to its peritoneal coat. The rest of the intestines usually escapes the action of the acid.

The *larynx* is very frequently acted on by the acid. There are yellow eschars, congestion and swelling of the mucous membrane, sometimes œdema of the glottis. The *trachea* may be inflamed and the *lungs* congested.

If the patient survives the first effects of the poison, the lesions of chronic inflammation, cicatrization, and contraction may be found at a later period.

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[1] *Woodman and Tidy*, "Forensic Medicine and Toxicology," ed. 1877, p 237.

The acid nitrate of mercury, if taken in a concentrated form into the stomach, may produce the same lesions as nitric acid.

HYDROCHLORIC ACID.

In fatal cases, death occurs on the average in about twenty-four hours. The lesions are in general similar to those produced by sulphuric and nitric acids, except that the eschars are usually of a whitish color at first, becoming, after a time, discolored and disintegrated. It is also more common to find false membranes on the inflamed surfaces.

OXALIC ACID.

In fatal cases, death may occur within ten minutes (in one case in three minutes) or may be delayed for two or three weeks. The period of death does not depend, as do in general the symptoms, upon the amount and concentration of the poison.

The mucous membrane of the *mouth, pharynx,* and *œsophagus* is usually white and shrivelled, and easily peeled off, and may be covered with brownish vomit from the stomach. The œsophagus may be much contracted. The *stomach* is usually contracted and contains a dark-brown, acid, mucous fluid. The mucous membrane of the stomach may be pale, soft, and easily detached, sometimes looking as if it had been boiled in water. Sometimes it is red and congested; sometimes blackened and gangrenous; sometimes peeled off in patches. Perforation is of rare occurrence. If life be prolonged, the whitened condition of the mucous membrane is succeeded by congestion and inflammation. The *small intestines* may be inflamed. Inflammation of the *pleura* and *peritoneum,* and congestion of the *lungs,* are of occasional occurrence. In some cases of death from oxalic acid there are no well-marked lesions.

Potassium oxalate produces the same lesions as oxalic acid.

TARTARIC ACID.

This acid is seldom used as a poison, but in large doses may prove fatal. The lesions in the cases observed were redness and inflammation of the mucous membrane of the gastro-intestinal canal.

POTASH, SODA, AND THEIR CARBONATES.

These substances are not commonly used as poisons with suicidal or homicidal intent, but may be taken by mistake. They may cause death in a few hours, or life may be prolonged for several weeks.

The mucous membrane of the *mouth, pharynx, œsophagus,* and *stomach* is softened, swollen, congested, and inflamed, or may be peeled off. It may be blackened from local changes in the blood. The mucous membrane of the *larynx* and *trachea* may also be swollen and inflamed.

If life is prolonged for some time, cicatrices and strictures of the *œsophagus* and *stomach* are apt to be produced as a result of the reparative inflammation.

AMMONIA.

The vapor of strong ammonia may cause death from inflammation of the larynx and air passages. The strong solution of ammonia produces lesions similar to those of potash and soda. The *larynx, trachea,* and *bronchi* are frequently inflamed, and may be covered with false membranes. Fatal inflammation of the rectum and colon has been produced by an enema of strong solution of ammonia.

POTASSIUM NITRATE.

Accidental poisoning sometimes occurs from large doses of this salt. In the observed cases, there were intense congestion and inflammation of the *stomach* and *intestines,* and in one case a small perforation of the stomach.

For the effects of several infrequently employed salts of the alkalies and alkaline earths, which for the most part produce simple inflammation of the gastro-intestinal canal, we refer to special works on toxicology.

PHOSPHORUS.

Poisoning by phosphorus is much more common in France and Germany than in this country. Some of the forms of rat poison. of which this is a frequent ingredient, and the ends of matches are common media for its administration. It is more often used with suicidal than homicidal intent.

The post-mortem appearances vary according to the length of time which elapses before death, which may be from a few hours to several months.

If death takes place in a few hours, the only lesions may be those produced by the direct local action of the poison. The mouth, pharynx, and œsophagus usually escape. The stomach may be only slightly reddened, or there may be patches of inflammation and erosion. The contents of the stomach are often mixed with blood, and may have the peculiar smell of phosphorus. There may be little bits of wood present when the poison has been taken from the heads of lucifer matches. It is said that the mucous membrane of the stomach may emit a phosphorescent light in the dark.

If death does not ensue until after several days, the lesions are more marked. The body is usually jaundiced. There may be ecchymosis beneath the pericardium, pleura, and peritoneum, in the lungs, the kidneys, the bladder, the uterus, the muscles, and the subcutaneous connective tissue, and bloody fluid in the visceral cavities.

The *heart and voluntary muscles*, the walls of the *blood-vessels*, and the endothelium of the air vesicles of the lungs may be in the condition of fatty degeneration. The blood is usually dark and fluid.

The *stomach* sometimes presents no very striking changes. There may be small circumscribed spots of inflammation, erosion, or gangrene, and occasionally perforation. The most constant change is a granular degeneration of the cells which fill the gastric follicles. In consequence of this, the mucous membrane appears thickened, opaque, of white, gray, or yellow color.

The *small intestine* appears normal or is congested.

The *liver* is found in different degrees of parenchymatous and fatty degeneration, and is often stained yellow from the jaundice. It is usually increased in size, and of a grayish, grayish-yellow, or light-yellow color, unless stained by the bile. Less frequently the centres of the acini are congested, or the entire liver is congested, or there are small hæmorrhages in the liver tissue. The liver may be soft, flabby, and smaller than normal. In the interstitial tissue of the liver and along the branches of the portal vein there may be marked infiltration with small spheroidal cells.

The *kidneys* often present parenchymatous and fatty degeneration of the epithelium. The *mesenteric glands* may be soft and swollen.

ARSENIC.

This poison is very frequently employed with suicidal intent. Death may occur in a longer or shorter time from the direct irritative effects of the poison upon the gastro-intestinal canal, with the symptoms which usually accompany the ingestion of irritant poisons; or it may occur with symptoms of collapse, or coma, or shock; or the symptoms may resemble those of cholera. The average time of death in acute fatal cases is about twenty hours, but death has occurred in twenty minutes, and has been prolonged for two or three weeks.

The *mouth*, *pharynx*, and *œsophagus* may be inflamed, but are more frequently unaltered. The *stomach* may be empty or contain mucus mixed with blood. The arsenic, in substance, may be found adherent to the mucous membrane or mixed with the contents of the organ. It has, in rare cases, been found incysted in the stomach in considerable quantity. When invisible to the naked eye, a microscopical examination of the stomach contents will not infrequently reveal characteristic crystals of arsenious acid or some of its compounds. The stomach may be contracted and its mucous membrane corrugated. The entire inner surface may be red and inflamed, or there may be patches or streaks of inflammation or deep congestion. The inflamed and congested patches may be thickened and covered with false membrane mixed with larger and smaller particles or masses of the poison. Ulceration, perforation, and gangrene are rare. Blood may be extravasated into the mucosa and submucosa, and with the congestion give the mucous membrane a very dark-red or brown appearance. Frequently the mucous membrane is studded with small petechiæ. Sometimes the arsenic is converted in the stomach into the yellow sulphide. There may be acute gastritis, even when the poison is absorbed by the skin or otherwise, and not introduced into the stomach. Taylor mentions a case in which the coats of the stomach were thickened and gelatinous, but not congested. The epithelium of the gastric glands may undergo granular and fatty degeneration.

The entire length of the *intestine* may be congested and inflamed, but the action of the poison does not usually extend beyond the duodenum. In some cases, the *solitary follicles*, *Peyer's patches*, and *mesenteric glands* are swollen. Inflammation of the *bladder* and *peritoneum*, and congestion and œdema of the *brain*, have been observed, but are neither frequent nor in any way characteristic.

Fatty degeneration of the *muscles*, *liver*, *kidneys*, *blood-vessels*, and *vesicular epithelium* of the *lungs* may be produced in arsenical poisoning.

Alterations in the spinal cord indicative of acute myelitis have been described by Popon as occurring in dogs poisoned with arsenious acid.[1]

The walls of the stomach and intestines and other parts of the body may be preserved from decomposition for a long time after death by arsenical poisoning.

It should always be borne in mind, in examining cases of suspected arsenical poisoning, that death may be produced by arsenic and its compounds without any appreciable lesions. While in general it may be said that in the cases in which no lesions are discovered death has been rapid, the death may be delayed in such cases until long after a period at which, in other cases, marked inflammatory changes have occurred.

Compounds of arsenic, such as the chloride and sulphide, and the arsenite (Scheele's green; Paris green), are sometimes used for suicidal purposes, and produce lesions similar to those of arsenious acid. Paris green is a favorite article in New York, particularly among Germans, for suicidal purposes. It is usually taken in considerable quantities, and is often found in the stomach after death.[2]

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[1] *Popon*, "Ueber die Veränderungen im Rückenmarke nach Vergiftung mit Arsen," etc. Virch. Arch., Bd. 93, p. 351.

[2] It is advisable, in cases of suspected arsenic poisoning, particularly if the body have lain for some time, as in exhumations, to preserve not only all of the internal organs entire for the chemist, but also portions of the muscles (back, thigh, arm, and

CORROSIVE SUBLIMATE.

The mucous membrane of the *mouth* and *throat* may be swollen, inflamed, or have a grayish-white appearance. The *œsophagus* may be swollen and white, or congested, or unaltered. The mucous membrane of the *stomach* is usually congested or inflamed, or there may be patches of softening, ulceration, or gangrene. Perforation is of rare occurrence. Small ecchymoses in the mucosa are not uncommon. Sometimes there is little or no change in the stomach. Sometimes the mucous membrane of the stomach is slate-colored from the deposition of metallic mercury from the decomposed salt. The *intestines* may appear normal, or there may be patches of congestion and ecchymosis.

The *larynx* and *trachea* may be congested. The *kidneys* may show parenchymatous and fatty degeneration of the epithelium.

LEAD.

The different preparations of lead may prove fatal, either from the immediate effect of large doses or from the gradual effects of repeated small doses. Although there may be marked symptoms during life, the post-mortem lesions are few and variable.

Large doses may produce acute gastritis, and sometimes a whitening of the mucous membrane. The intestines are generally contracted, and there may be fatty degeneration of the renal epithelium; very frequently there are no appreciable lesions.

In chronic lead poisoning, the intestines may be contracted, the voluntary muscles flabby and light-colored, or partially replaced by connective tissue, and there may be chronic meningitis.

COPPER.

Acute poisoning by salts of copper is not very common, but it is of occasional accidental occurrence, and the salts are infrequently used with suicidal intent. The sulphate and acetate are the most important salts in this respect. Soluble salts of copper may be formed in the use of copper cooking utensils, and accidents most frequently occur in this way.

The post-mortem appearances are somewhat variable. The *pharynx*

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abdomen), and also one of the long bones, preferably the femur, since arsenious acid and its compounds are quite diffusible, and may be present in proportionately larger quantity in other parts than in the gastro-intestinal canal. It is desirable to save the whole of the internal organs, and to weigh the muscle and bones as well as the whole body at the autopsy, in order that the calculations of the chemist, in case arsenic be found, may rest upon a definite basis, and be as little as possible dependent upon estimates, whose value may be questioned by lawyers should the case come into the courts.

and *œsophagus* may be somewhat inflamed or unchanged. The mucous membrane of the *stomach* and *intestines* may be inflamed, ulcerated, or gangrenous, and perforation and peritonitis may occur. The mucous membrane may have a diffuse greenish color, or particles of the salt may be found adhering to it.

TARTAR EMETIC.

This preparation of antimony may prove fatal when administered in a single large dose or in repeated small doses. The post-mortem lesions are not constant. In cases of chronic poisoning there are usually no appreciable lesions.

In cases of acute poisoning there may be evidence of acute inflammation of the *œsophagus, stomach, intestines,* and *peritoneum.* Sometimes the stomach exhibits no lesions, while the intestine is involved. The *larynx* and *lungs* may be deeply congested.

VEGETABLE IRRITANTS.

Aloes, colocynth, gamboge, jalap, scammony, savin, croton oil, colchicum, veratria, hellebore, elaterium, and *turpentine.*

All these drugs may produce poisonous effects. The post-mortem lesions are congestion, inflammation, and sometimes ulceration of the gastro-intestinal mucous membrane; but these lesions are sometimes present and sometimes absent.

CANTHARIDES.

This substance may be given in powder or tincture. The entire length or only a portion of the *alimentary canal* may be congested or inflamed. There may be patches of gangrene of the mucous membrane of the *stomach.* When the poison was taken in substance, a microscopical examination of the contents of the alimentary canal or of the mucous membrane may reveal the glistening green and gold particles of the fly.

The *kidneys, ureters,* and *bladder* may be congested and inflamed. There is sometimes congestion of the *brain* and its membranes.

OPIUM.

The post-mortem appearances in persons who have died from opium poisoning are inconstant and not characteristic. Congestion of the *brain* and its membranes, with serous effusion in the membranes and ventricles, and congestion of the lungs, are changes occasionally seen, but they are frequently entirely absent, and when present are not characteristic of death from this poison.

POISONOUS FUNGI.

The action of these substances varies greatly, and the post-mortem appearances are inconstant and not characteristic. In general, when any lesions are present, they are those of gastro-intestinal irritation or of venous congestion, or both.

Microscopical examination may reveal characteristic fragments of fungi in the contents of the alimentary canal.

HYDROCYANIC ACID.

This poison in fatal doses may destroy life in a very short time. The post-mortem appearances are inconstant and not characteristic. The skin may be livid and the muscles contracted. The *stomach* may be congested or normal. The most frequent internal appearances are those of general venous congestion. Under favorable conditions the odor of prussic acid may be detected in the stomach or blood or brain or other parts of the body. It may be absent in the stomach and present in other parts of the body. If the patient have lived for some time, the odor may be absent altogether.

Cyanide of potassium may produce the same lesions as prussic acid, and there is the same inconstancy in their occurrence.

Nitrobenzole.—This substance produces general venous congestion, and the odor of the oil of bitter almonds may be more or less well marked in the body after death.

CARBOLIC ACID.

When this poison is taken into the stomach, the mucous membrane of the *mouth*, *œsophagus*, and *stomach* may be white. corrugated, and partially detached in patches, and the edges of the affected parts may be hyperæmic or there may be patches of extravasation. Brownish shrunken patches may be present about the mouth. The *brain* and *meninges* may be congested. There may be congestion and œdema of the *lungs*, and congestion of the *liver* and *spleen*. The blood is usually dark and fluid. The *urine* is usually of a dark or greenish color. The odor of the poison may be evident in the body and in the urine.

ALCOHOL.

The different preparations of alcohol, when taken in concentrated form or in large quantities, sometimes produce sudden coma and death in from half an hour to several hours. In acute poisoning, if death have followed soon after the ingestion of the poison, the body may resist decomposition for an unusual length of time. The stomach and tissues may even have a more or less well-marked alcoholic odor. The *stomach*, and even the *œsophagus* and *duodenum*, may be of a deep-red color. There may be punctiform ecchymoses in the gastric mucous membrane. In many cases, the stomach is apparently quite normal. There is apt to be venous congestion in some of the internal organs, but this is not constant. There is frequently congestion and sometimes extravasation of blood in the *brain* and its *membranes*, and œdema of the membranes or of the brain substance, or both. There may be a serous effusion in the ventricles of the brain. The bladder is frequently distended with urine, as in other cases in which death is preceded by a period of unconsciousness.

Chronic alcoholic poisoning is of a different nature. The subjects of it may die from some other disease, or they die after a debauch without anything else to account for their death. In the latter case there may be *delirium tremens*, or the patient dies exhausted and comatose. Chronic alcoholism is not infrequently mistaken clinically for meningitis. The post-mortem lesions are sometimes marked, sometimes absent. There may be chronic pachymeningitis, resulting in thickening of the *dura mater* and its close adherence to the skull. The *pia mater* may be thickened and œdematous. The *brain* may be normal or œdematous or atrophied. The *lungs* are frequently congested. The heart may be thickly covered with fat, and its walls may be flabby and fatty. The *stomach* frequently presents the lesions of chronic gastritis. The *liver* may be cirrhotic, with or without fatty infiltration. The *kidneys* may present the lesions of parenchymatous or fatty degeneration or of chronic diffuse nephritis.

It should always be remembered, however, that all or a part of the above lesions may be absent in the bodies of drunkards, and, furthermore, that the same lesions may be due to other causes.

CHLOROFORM.

Chloroform may cause death when it is taken in fluid form into the stomach or when inhaled. Death from swallowing liquid chloroform is rare, and its immediate cause is usually uncertain. The post-mortem changes are variable; sometimes there are no lesions. In some cases there is simple reddening of the gastric mucous membrane; occasionally there is acute gastritis or ulceration of the mucous membrane. The odor of chloroform may or may not be evident. Discoloration and softening of the mucous membrane of the pharynx, œsophagus, and duodenum have been observed. There may be general venous congestion; the heart may be flabby. Bubbles of gas have been frequently seen in the blood, but this is not characteristic. Death from inhalation of chloroform is a not infrequent accident in surgical practice. After death from inhalation, the results of the examination are usually quite negative.

ETHER.

The inhalation of ether occasionally causes death. The post-mortem examination is negative. The ingestion of fluid ether may induce inflammation of the stomach. The odor of ether may be perceptible if the autopsy is made soon after death.

CHLORAL HYDRATE.

There are no characteristic post-mortem appearances after death by chloral. Hyperæmia of the brain, and the odor of the drug, have been noticed.

STRYCHNIA—NUX VOMICA.

The post-mortem appearances after poisoning by these drugs are not characteristic, and are inconstant. The body is usually relaxed at the time of death, but the rigor mortis usually comes on early and remains long. There may be congestion of the *brain* and *spinal cord,* and sometimes of the *lungs* and *stomach.*

CONIUM, ACONITE, BELLADONNA, LOBELIA INFLATA, DIGITALIS, STRAMONIUM.

These vegetable poisons are administered in their natural form of leaves, berries, and roots, or in tinctures, infusions, and extracts, or in the form of their active alkaloid principles.

If the leaves, berries, or seeds are given, they may be detected in the contents of the stomach by microscopical examination. Otherwise the results of autopsies are not characteristic.

The *brain* and its membranes, and the lungs, may be congested. The *stomach* may present patches of congestion, inflammation, and extravasation, or its entire mucous coat may be inflamed, or it may appear normal.

Microscopical examination of the contents of the alimentary canal may reveal characteristic seeds or fragments of leaves.[1]

CARBONIC OXIDE.

This is one of the gases formed in the burning of charcoal, and forms one of the ingredients of illuminating gas. The most characteristic post-mortem appearance is the cherry-red color of the *blood,* and of the tissues and viscera which contain blood. The presence of carbonic acid in the gas may obscure the bright red of the carbonic oxide by the dark color which it induces in the blood.

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[1] Consult *Guy and Ferrier,* "Forensic Medicine," p. 534.

CARBONIC ACID.

The lesions are essentially those of asphyxia, but the brain is said to be more frequently congested than in asphyxia by simple obstruction of respiration.

For a more detailed consideration of poisons, their effects, modes of detection, etc., consult *Taylor* on Poisons; *Maschka's* "Handbuch der gerichtlichen Medicin," Bd. 2; *Woodman and Tidy*, "Forensic Medicine." *Wormley's* "Micro-chemistry of Poisons" contains a series of good plates of the microscopical appearance of various forms of crystals of poisonous substances.

Lesser's "Atlas der gerichtlichen Medicin" contains a series of fine colored plates showing the appearance of the stomach after the action of various poisons. The small work of *Guy and Ferrier*, on "Forensic Medicine," contains in very compact and reliable form much information on the general subjects treated in the foregoing section.

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